EXHIBIT G

Scott D. Phillips, MD, FACP, FACMT, FAACT
Declaration and Report

IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TENNESSEE
AT KNOXVILLE

GREG ADKISSON, et al., Plaintiffs, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No. 3:13-CV-505-TAV-HBG *Lead case consolidated with*
KEVIN THOMPSON, ET AL., Plaintiffs, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No. 3:13-CV-666-TAV-HBG *as consolidated with*
JOE CUNNINGHAM, et al, Plaintiffs, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No. 3:14-CV-20-TAV-HBG
BILL ROSE, Plaintiff, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No. 3:15-CV-17-TAV-HBG
CRAIG WILKINSON, ET AL., Plaintiffs, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No.: 3:15-CV-274-TAV-HBG
ANGIE SHELTON, as wife and next of kin on behalf of Mike Shelton, et al., Plaintiffs, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No.: 3:15-CV-420-TAV-HBG

JOHNNY CHURCH, Plaintiff, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No.: 3:15-CV-460-TAV-HBG
DONALD R. VANGUILDER, JR., Plaintiff, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No. 3:15-CV-462-TAV-HBG
JUDY IVENS, as sister and next of kin, on behalf of JEAN NANCE, deceased, Plaintiff, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No. 3:16-CV-635-TAV-HBG
PAUL RANDY FARROW, Plaintiff, v. JACOBS ENGINEERING GROUP, INC., Defendant.	)	No. 3:16-CV-636-TAV-HBG

DECLARATION OF SCOTT D. PHILLIPS, MD, FACP, FACMT, FAACT

I, Dr. Scott D. Phillips, a board certified medical toxicologist, declare as follows:

1. My name is Dr. Scott D. Phillips. I am over the age of 21, I have the use of reason, I understand the nature of my oath, and I am capable of making this declaration. The facts stated in this declaration are true and correct based upon my personal knowledge.

2. I am currently employed as a Partner at NewFields, LLC, an environmental consulting firm. I am an Associate Professor at the University of Colorado, Department of Medicine, Division of Clinical Pharmacology and Toxicology. I am a faculty member at the Rocky

Mountain Poison & Drug Center and the Washington Poison Center. I am board certified in both internal medicine and medical toxicology and am licensed in the states of Colorado and Washington.

3. Counsel for Jacobs Engineering Group, Inc. retained me to prepare an expert report.

4. I prepared the attached expert report, dated August 16, 2017, that I hereby adopt in full as if it were stated in this declaration in its entirety.

I declare under penalty of perjury that the foregoing is true and correct.

October 5th, 2017

Dr. Scott D. Phillips

NewFields

PO Box 13250
Burton, WA 98013
303-815-1960

REPORT PREPARED BY

Scott D. Phillips, MD, FACP, FACMT, FAACT

FOR
Adkisson et al v. Jacobs Engineering Group, Inc.

16 August 2017

INTRODUCTION

I hold the following opinions and conclusions in this report to a reasonable degree of medical and toxicological probability (certainty). Should further information become available, I reserve the right to amend my report or opinions. The basis of my opinions is put forth in the body of this report.

SUMMARY OF OPINIONS AS TO TOXICOLOGY

1. Plaintiffs have not been exposed at Kingston Fossil Plant to levels of fly ash sufficient to cause illness.

2. The large fly ash particles are mostly deposited in the upper (non-gas exchanging) area of the lung and respiratory tract.

3. Metals are bound to the fly ash particles and are not dissolving out of the particles and into the body to cause illness.

4. There are no elevated metals levels in plaintiffs, indicating no dissolution of the fly ash particles.

5. Plaintiffs have not performed a differential diagnosis nor followed an accepted methodology for causation analysis.

6. Plaintiffs do not have illness from exposure to fly ash at the Kingston Fossil Plant clean-up site.

STATEMENT OF QUALIFICATIONS

I am Scott D. Phillips, Partner at NewFields, LLC (an environmental consulting firm). I am an Associate Professor, at the University of Colorado, Department of Medicine, Division of Clinical Pharmacology and Toxicology. I am a faculty member at the Rocky Mountain Poison & Drug Center and the Washington Poison Center. I am board certified in both internal medicine and medical toxicology and am licensed in the states of Colorado and Washington.

The American Board of Medical Specialties (ABMS) is the medical oversight organization that 1) is the umbrella authority of all U.S. medical boards, 2) defines all specialties and sub-specialties that are officially recognized in the United States, and 3) certifies, "a physician's exceptional expertise in a particular specialty and/or sub-specialty of medical practice." The ABMS classifies medical toxicology as a sub-specialty.

Medical toxicology is a scientific discipline concerned with the evaluation, diagnosis, and

treatment of adverse effects of chemical substances, including pharmaceuticals, on living systems. Fundamental to the sub-specialty is that medical toxicologists must routinely perform an assessment of whether there exists a causal link between an exposure and an adverse effect. Such an analysis requires the application of proper and generally accepted scientific methodologies.

My medical toxicology training consisted of a two-year post-doctoral fellowship at the Rocky Mountain Poison and Drug Center and the University of Colorado Health Sciences Center in Denver, Colorado. This training culminated with my passing the required board certifying examination. The American Boards of Preventive Medicine, Pediatrics and Emergency Medicine as the Sub-Specialty Sub-Board on Medical Toxicology jointly support the board certification. I initially certified in 1995, the first year the board was given, and I re-certified in 2005 and 2016. Moreover, physicians who are board certified in internal medicine are eligible for the joint medical toxicology boards through the American Board of Emergency Medicine pathway.

I am a member of the American Academy of Clinical Toxicology, and the American College of Medical Toxicology. I have served on both governmental and non-governmental advisory panels and committees. I have been awarded research grants.

I am an Associate Clinical Professor in the Department of Medicine, Division of Clinical Pharmacology and Toxicology at the University of Colorado. Additionally, I am an attending physician with the Rocky Mountain Poison and Drug Center, and the Washington Poison Center. I have a clinical medical practice in Washington.

In my teaching role, I provide both didactic lectures and bedside clinical teaching in medical toxicology and internal medicine to nurses, medical students, interns, residents, and fellows in medical toxicology sub-specialty training.

My clinical practice comprises treating toxicology patients in emergency departments, intensive care units, the general hospital, and in the outpatient setting. During my clinical practice, I routinely evaluate and treat patients with all types of clinical illnesses including, but not limited to, neurological, cardiovascular, pulmonary, oncology and metabolic disorders. I have over 25 years of experience related to the investigation and evaluation of exposure to potentially hazardous materials. I have both published and presented extensively in the fields of toxicology, occupational environmental medicine and public health and have co-edited several books on Occupational Medicine and Medical Toxicology. I routinely teach toxicology to physicians and lecture internationally.

Throughout my career, I have served in various capacities in the American Academy of Clinical Toxicology (AACT) and other major societies dealing with clinical toxicology. I have been elected to the Board of Directors of the AACT and have served on many committees of that organization. The AACT is the largest organization in the world devoted to clinical aspects of toxicology and has among its members virtually all medical toxicologists and poison control center directors from the United States, Canada, and many other countries. In addition, many regulators,

policy makers, and scientists with an interest in clinical toxicology are members of AACT.

I have authored and published approximately 200 scientific articles, numerous peer-reviewed papers, letters to the editors, editorials, book chapters, and abstracts, virtually all of them related to clinical toxicology. I also have served as an editor of several major toxicology texts.

A complete statement of my publications, academic appointments and qualifications is described in my attached *curriculum vitae*.

SCOPE

The plaintiffs claim damages resulting from exposure to fly ash during the clean-up of the Kingston Dredge Cell Incident Recovery Program Project at the TVA Kingston Fossil Fuel Plant located in Kingston, Tennessee (the "KIF fly ash recovery project").

Attorneys representing Jacobs Engineering Inc. have requested that I offer opinions regarding (1) toxicology as it relates to exposure to fly ash and its components, and (2) the potential health effects related to said exposure, (3) causation analysis of health claims and fly ash, and (4) usefulness of medical monitoring program.

MATERIALS REVIEWED

Regarding this case, I have reviewed the following documents:

- Affidavit of Jack Howard
- Affidavits of Thomas Bock
- Affidavit of Chris Eich
- Affidavits of John Cox
- Affidavit of Kevin Thompson
- Affidavits of Ansol Clark
- Affidavit of Ronald Bledsoe
- Affidavits of Stan Hill
- Affidavits of Glenn Knight
- Affidavit of Kenneth Wright
- Affidavit of Doug Bledsoe
- Affidavit of Billy Gibson
- Affidavit of Mike McCarthy
- Affidavit of Jimmy Roberts
- Affidavit of Dan Cody
- Affidavit of Timothy Gibson
- Affidavit of Brian Summers
- Affidavit of Fred Chris Jones
- Affidavit of William Hedgecoth
- Affidavit of Johnny O. Church
- Affidavit of Ernie Turpin
- Affidavit of David Johnson
- Affidavit of Joe Pursiful
- Affidavit of Roy Edmonds

- Affidavit of Frankie Norris
- Affidavit of Harvey Bass
- Affidavit of Leonard Ronald Bledsoe
- Affidavit of Craig Wilkinson
- Affidavit of Carl Booker
- Declaration of Jack Howard
- Declaration of Thomas Heffernan
- Deposition of Tom Heffernan
- Deposition of John Parker
- Deposition of Jamie Keith
- Deposition of Ansol Clark
- Deposition of Billy Joe Gibson
- Deposition of Bradford Green
- Deposition of Brian Thacker
- Deposition of Steven L. Cherry
- Deposition of Dwayne Rushing
- Deposition of Gary MacDonald
- Deposition of Gene Meredith
- Deposition of Jeff Brewer
- Deposition of John Cox
- Deposition of Johnny Church
- Deposition of Mike McCarthy
- Deposition of Stanley J. Hill, Jr.
- Deposition of Thomas Bock
- Jacobs TVA Contract, Part I
- Industrial Hygiene Monitoring Plan R1 April 2012
- CP SEG Summary
- IH Sampling Report Kingston Fossil Ash Project 8728
- KIF List of Training by Individual 01
- Orientation Handout
- Position on Steel Toe
- Sample Numbers by analyte
- SWSH Plan Booklet
- Legal Filings
- Ensafe Plaintiff Monitoring
- Employee file Duration of Employment on Project
- Plaintiffs Responses to Interrogatories
- Site Dust Control and Air Monitoring Plan
- EPA-AO-TVA Corrective Action Plan
- TVA MSDS Class "F" fly Ash No.-001, 6/2001
- ATSDR PHA TVA Kingston Fossil Plant 9-7-2010neur
- Site Wide Safety and Health Plan for TVA Kingston Fossil Plant Ash Release Response 2010
- TetraTech Report to USEPA 2009
- TVA and EPA Kinston Ash Recovery Project Completion Report
- Photos from Mike McCarthy
- Plaintiffs Expert Disclosures
- Plaintiffs Recording
- TVA Project Closeout USEPA

- Public Health Assessment of the Kingston ash spill (PHA) prepared by the Tennessee Department of Health (TDH) Environmental Epidemiology Program

Additionally, I have examined site-related documents, consulted appropriate regulatory guidance documents, and performed an independent literature review and analysis of the relevant medical and toxicological literature. A bibliography of reference material used in preparation of this report is provided. If other relevant materials become available, I reserve the right to amend or modify my opinions.

SUMMARY OF OPINIONS AS TO EXPERT DISCLOSURES

1. In reaching their opinions (outlined in their designations) regarding plaintiffs' exposures and risks due to fly ash exposure, plaintiffs' experts did not use accepted scientific methodology. Plaintiff's fatal flaws include: no consideration of exposure, no indication of a specific toxin, no differential diagnosis or alternative etiology assessment, no discussion of dose or dose-response as it pertains to the measurement of chemicals of potential concern (COPCs) . Plaintiffs' claims for general causation are not met.
2. No evidence exists that plaintiffs were exposed to clinically significant concentrations of fly ash, and so therefore no medical basis exists regarding fear of developing injury or disease.
3. No evidence exists that plaintiffs were exposed to harmful levels of fly ash or any other chemicals. This is based on air sampling conducted at the KIF site. This is also based on plaintiffs' limited sampling of blood and urine to assess levels of chemicals including, but not limited to, arsenic, mercury, lead, cobalt, thallium and cadmium.
4. In the absence of documented evidence of elevated exposures to fly ash, there is no scientific basis for recommending medical monitoring.

BACKGROUND INFORMATION

Methods Scientists Use to Infer Causation

Health regulatory agencies throughout the world concur on conceptual framework and methodology for assessing potential toxicological risks. This framework is described in many textbooks of occupational & environmental medicine and toxicology (Sullivan 2001; Klaassen 2013) and integrated into international regulatory and other guidance documents published by (among many others) the National Research Council (NRC 1983; NRC 1991), U.S. Environmental Protection Agency (EPA 1989, 1992, 2011), the World Health Organization (WHO 1999, 2000), the Agency for Toxic and Materials (ASTM 2010a&b). According to the consensus, a scientifically defensible conclusion that a chemical exposure caused a given health effect, or that any individual or group is at significantly increased risk of adverse effects from a certain chemical

exposure, requires rigorous elucidation of each element of the logical sequence:

Source → Exposure → Dose → Potential Health Effects(s)

Following this sequence, an investigation to determine that an illness or health effect has been or could be caused by exposure to chemicals must proceed in a logical fashion that (1) establishes the presence of a complete exposure pathway[1] linking a chemical source(s) to the human receptor, (2) estimates the concentration(s) of any source-related chemical(s) under investigation at the receptor's location via measurements or modeling over the exposure period, (3) calculates or measures the dose received by the individual(s) at the exposure point, and (4) characterizes the potential health effects of the chemical(s) under investigation based upon the route of exposure and chemical-specific dose-response relationship(s).

Without sufficient exposure concentration resulting in a quantifiable dose, it is scientifically and medically incorrect to assume a significant health effect(s) occurred. Exposure is an environmental concentration of a substance and opportunity for contact with the substance through swallowing, breathing, or touching the skin or eyes. Dose is the concentration of a substance that is in or on the body available to interact with receptors. (Letz 2014)

BASIC PRINCIPLES OF TOXICOLOGY

Toxicology is the field of science that investigates and describes whether and how exposure to environmental factors causes adverse (toxic) effects in organisms, including humans. Exposure is a term describing the first tenet of toxicology; in a biological system, the effect, i.e., the hazard it poses, of any chemical is determined by the magnitude and timing and duration of exposure (dose rate) and exposure route (ingestion, inhalation, dermal absorption) – not simply by exposure itself. The 16th Century physician Paracelsus (Klaassen 2013), famously articulated the central dose-response principle.

> "What is there that is not poison? All things are poison, and nothing is without poison; the dose alone makes the thing no poison."

Simply, the toxic effects of a chemical depend on dose (how much), frequency of exposure (how often), duration of exposure (how long), and the route by which the chemical enters the body. Dose-dependence is a general characteristic of toxicological responses, including all forms of adverse effects. Ignoring this essential relationship does not satisfy the Source → Exposure → Dose → Health Effect(s) paradigm, and thus violates the most fundamental tenet of toxicology:

[1] Defined as "the course a chemical or physical agent takes from a source to an exposed organism." A complete exposure pathway includes a source or release from a source, an environmental transport/exposure medium (or media), an exposure point (location of potential contact between an organism and a chemical or physical agent), and an exposure route (*i.e.*, ingestion, inhalation, dermal contact) (EPA 1989).

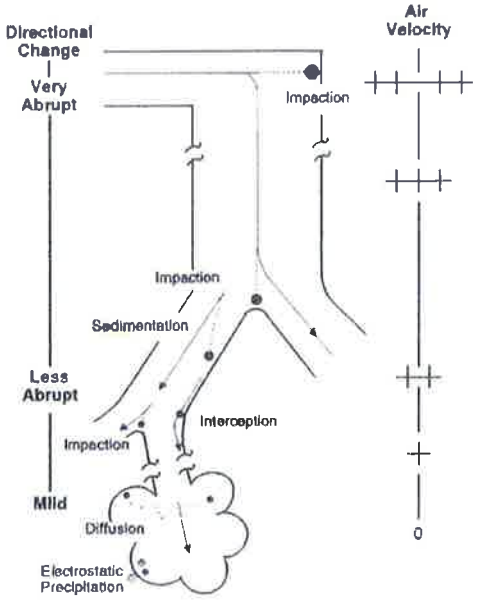
the dose makes the poison. Appraisal of adverse toxicological risk probability requires knowledge of (1) the chemicals that posed intrinsic hazard(s), and (2) the dose or concentration to which an individual is exposed, and (3) illness caused by absorbed dose that is demonstrated in the medical literature. Moreover, for all individuals, other reasonable causes of claimed health effect(s) must be ruled out before chemical causation can definitively be ruled in.

The second tenet of toxicology is that individual chemicals exert specific toxic effects that are determined by their unique chemical structures (e.g., Rozman and Doull 2000; Goldstein and Gallo 2001). Involved in the interaction between a chemical and an organism, there are two fundamental elements: (1) what the organism does to the chemical (pharmacokinetics), and (2) what the chemical does to the organism (pharmacodynamics). Both elements are dependent on the specific characteristics of both the chemical and the organism. In order to assess specific causation for exposed individuals, all of these considerations, i.e., dose, exposure frequency and duration, pharmacokinetics, and pharmacodynamics, are critical.

There are four routes by which a substance can enter the body: 1) inhalation; 2) skin absorption (including eyes and mucus membranes); 3) ingestion; and 4) parenteral. For chemicals in the form of vapors, gases, mists, or particulates, inhalation is the major route of entry. Once inhaled, chemicals are either exhaled or deposited in the respiratory tract (see following figure). For the chemical to diffuse into the blood through the lung-blood boundary, dissolution must occur. In the case of fly ash exposure, the potential route of exposure is inhalation.

GEOMETRIC SIZE DEPOSITION

If inhaled, chemicals are either exhaled or deposited in the respiratory tract (see following figures). For coarse particles, impaction is a significant mechanism by which the particles may be deposited.

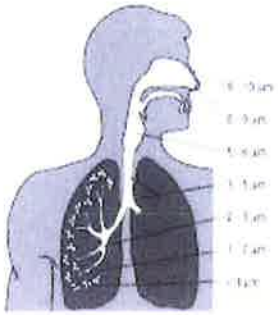


Coarse particles have a greater mass and velocity in the air column, and impact in the upper airway and early branching of the bronchi. As air flow velocity decreases and more branching continues, there is more time for gravitational forces (sedimentation) to settle the particle in the distal (deeper) airway. Sedimentation occurs because of the influence of the gravity on the particles. The following diagram illustrates particle influence by air flow (velocity) and lung branching. (EPA 1994)

The lymphatics and mucocillary ladder remove particles deposited in the lung. (ATSDR, 2005).

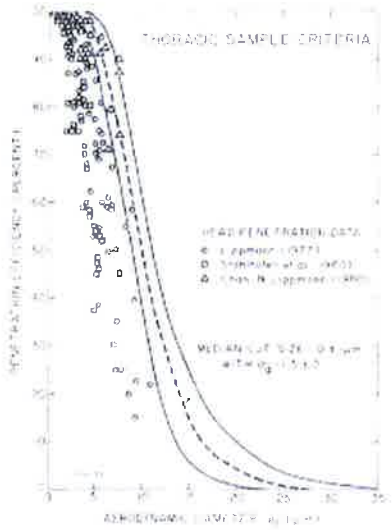
Air pollution typically is divided into the gaseous phase (sulfur and nitrogen oxides, ozone, and etc.) and particulate phase (dusts composed of organic and inorganic mater). (Wilson 1997) The particulate phase frequently is reported based on particle size called coarse particles and also called PM_{10}. The PM_{10} is a measure of particles that are 10 microns and smaller. Fine particles are a portion of the PM_{10} that are < 2.5 microns ($PM_{2.5}$). Microns (micrometers) is a unit of length. A 2.5 micrometer particle is the same as 0.00025 centimeters. A microscope is required to see fine particles. For comparison, a human hair is between 50-150 microns. Fine particles are considered the most important, because they can penetrate deeper into the breathing tubes of the lung.

Coarse particles (PM10) are larger and heavy and collide with the tissue in the upper respiratory tract. Coarse particles are more like a thrown baseball, while fine particles (PM2.5) behave more like smoke that follows air currents. The smaller the particle, the deeper it can penetrate the lung. The following figure illustrates this relationship.



As smaller particles penetrate deeper into the lung tissue, high levels of fine particles can cause inflammation or bronchoconstriction and can be absorbed.

The general site of mass particle deposition in the airway is illustrated in the following figure.



Based on aerodynamic diameter, finer particles have greater ability to penetrate deeper into the airway and gas exchange regions of the lung. If deposited in the lung, dissolution may occur. Dissolution of a particle means that the bonds holding the constituent elements of the particle together are broken. If dissolution of the particle occurs, the constituent elements may, but are not necessarily, available for absorption by the body. The required environment for dissolution to occur is addressed in more detail below. (*See infra, Toxicity of Chemicals of Concern)*

OVERVIEW OF THE HEALTH RISK ASSESSMENT

The basic principles of toxicology underlie the process of quantitative human health risk assessment that has been developed by regulatory authorities such as EPA, ATSDR, WHO, Health Canada, and the Netherlands National Institute for Public Health (RIVM) over the past three decades. The risk assessment process consists of four basic steps (*e.g.*, NRC 1983; EPA 1989; WHO 1999):

- **Hazard Assessment** – The determination of whether a chemical can cause health effects under any exposure conditions.
- **Dose-Response Assessment** – The determination of the relationship between exposure (dose) and the probability of occurrence of these adverse health effects (response).
- **Exposure Assessment** – The determination of the extent of human exposure.
- **Risk Characterization** – The characterization of toxicological risk requires the integration of objective data and the use of current science-based inferences regarding hazard, mode of action, dose, dose-response, and exposure. A description of the nature and often the potential magnitude of human risk, including attendant uncertainties must be considered.

The purpose of risk assessment is to provide a consistent, rational methodology for setting exposure standards and environmental cleanup goals and prioritizing sites for remedial action based on the likelihood of potential adverse health effects. However, it is important to recognize

that risk assessment calculations do not apply to specific individuals, and cannot be used to predict or explain individual health conditions. This process may rely on extrapolation from high-dose animal studies and conservative policy choices are intended to ensure that potential exposures and risks to humans will be consistently **overestimated** rather than underestimated. Thus, regulatory toxicity criteria (and risk-based screening levels based upon them) represent levels at which no adverse effects are expected to occur – "no effects" levels with a substantial margin of safety – rather than bright lines above which adverse effects occur.

Exposure Assessment

A health complaint alone cannot be used to determine source, exposure, or dose. According to the universally accepted paradigm, for either local or systemic toxicity to occur, there must be a complete exposure pathway between the chemical and the "receptor" (exposed individual or population). The actual dose received by an individual depends on numerous factors, including chemical concentration, frequency and duration of contact, route of exposure, and a variety of medical and other susceptibility factors specific to the individual. For purposes of risk assessment, conservative exposure estimates are calculated based on (1) estimated chemical concentrations in potential "exposure media" (environmental media [e.g., soil, water, air] with which people might come into regular contact), and (2) assumptions regarding rates of contact with these media. Conservative default exposure assumptions intended to ensure that potential exposures will be consistently **overestimated** rather than underestimated are provided in EPA guidance[2] (EPA 1992, 1997b, 2009).

For the first 2-3 months of the remediation, OSHA PEL values were used. However, TVA decided to use an even lower value corresponding to one-half of the OSHA PEL. This resulted in an even more protective environment.

Hazard Identification and Dose-Response Assessment (Toxicity Assessment)

It is critical to clearly distinguish between the concepts of "hazard" and "risk". The term "hazard" refers to the effect(s) a chemical potentially causes, without regard to the dose or exposure. "Risk" is the likelihood that under defined exposure conditions, an adverse health effect will occur.

Hazard identification identifies the adverse health effects caused by a chemical, while dose-response assessment characterizes the relationship between exposure or dose and the incidence and severity of effects. Together, they constitute toxicity assessment.

Risk Characterization

Risk characterization is the culmination of the risk assessment process, combining the results of the dose-response (toxicity) and exposure assessments to provide numerical estimates of potential health risks associated with specific potentially complete exposure pathways. Risk characterization also considers the nature and weight of evidence supporting these risk estimates,

[2] The EPA Guidance was a general toxicity assessment document and not specific to Kingston.

as well as the sources and impacts of key uncertainties attending them (*e.g.*, EPA 1989, 2000, 2009b).

TOXICITY OF CHEMICALS OF CONCERN

Fly Ash

Fly ash from burning coal is an agglomeration of materials, mostly metals, bonded to silica. The particles can vary in size from sub-micron to tens of microns in diameter. (Natusch 1974, Coles 1979) Particle diameters and chemical configurations are determined by the geographical origin of coal, the pyrolysis process and the cooling phase. The metals generally associated with coal sourced fly ash may include: aluminum, antimony, arsenic, barium, beryllium, boron, cadmium, calcium, chloride, chromium, cobalt, copper, fluoride, iron, mercury, magnesium, manganese, molybdenum, nickel, lead, phosphorus, potassium, selenium, silicon, sodium, strontium, sulfur, tin, titanium, vanadium, zinc, and radionuclides (e.g., radium-226) (Iyer 2002, USEPA 2009)

In humans, fly ash particle dissolution is determined by size, mineral, solubility and pH (the concentration of hydroxyl ions). (Kim 2003, Dudas 1991) For example, arsenic requires an acidic pH (pH less than 7) for dissolution that would allow it to be absorbed. Measuring arsenic in body fluids also requires speciation to determine if arsenic is of a potentially harmful form. Selenium requires a very high pH (pH greater than 7) for dissolution. (Lin 2013). The pH in the lungs is not acidic enough or basic enough for the constituent ash conglomerate to dissolve, *i.e. metals* breaking apart from, the ash particles. Since dissolution of the constituent elements could not have occurred, absorption of the constituent elements into the blood could not have occurred.

The following is a table of metal concentrations in Plaintiffs. There are no elevated levels that would suggest dissolution has occurred. This undercuts Plaintiffs' claims. Dr. Rea and Terry did not discuss these findings.

Plaintiffs Metal & Testosterone Tests

Test	Brewer, Jeffrey D (10-13-2011)	Clark, Ansel (5-28-2013)	Gibson, Billy Joe (4-12-2013)	Norris, Frankie E (5-2-2013)	Norris, Frankie E (4-30-2013)	Ramey, Ralph G (5-6-2013)	Summers, Brian (5-23-2013)	Thompson, Kevin (5-24-2013)	Williams, Jason (4-12-2011)
Lab Name	Quest Diagnostics	Quest Diagnostics	American Esoteric Lab	Personal Physician Care Lab	Personal Physician Care Lab	LabCorp	LabCorp	LabCorp	Nichols Institute
Pb (mcg/dl) Bld [NI <10]	<3		<2	1	<3.3*	ND			1.6
Hg (mcg/L) Bld [NI<10]	<5		<4	ND		ND		ND	
As (mcg/L) Bld [NI <23]	<10		<3	7		6		4	
As 24-Hr (mcg/L) [≤ 80]		20					51 [0-50]		
Pb 24-Hr (mcg/L) [<80]		<10							
Hg 24-Hr (mcg/L) [≤20]		<4							
As Rd Urine [0-50]		12.4					19		BDL
Pb Rd Urine [0-49]		ND					ND		
Hg Rd Urine [0-19]		ND					ND		BDL
Cd Rd Urine		ND							BDL
Co Rd Urine		0.5							
Th Rd Urine		ND							
Cd blood (mcg/L) [0.0-1.2]						ND	ND [ND]		
Arsenic Inorg U [0-19]							ND		
ZPP [< 41 mcg/dl]									17
Testosterone (ng/dl) [348-1197]			189 [241-827]			351 ng/dL [348-1197]		612 ng/dL [348-1187]	
Testosterone-Free			6.4 pg/ml [6.8-21.5] Warde Medical Lab						
	ND = Not Detected	ND = Not Detected		ND = Not Detected	* Ped Lead test (range 3.3-10.0)	ND = Not Detected	ND = Not Detected	ND = Not Detected	BDL= Below Detectable Limits

This is consistent with the findings that the outer layer of the fly ash particle has less silica and aluminum oxide as compared to the levels of potassium, sodium, calcium, magnesium and iron. (Brouwers 2003)

At the Kingston Fossil site, measured air dust levels were below OSHA levels and site safety levels. (PHA, Page 16) Therefore, with low air levels, there would be low exposure concentrations, and limited deposition in lungs. Even if fly ash were to penetrate deep into the lungs, the lung is not acidic enough to cause arsenic dissolution. (Dressen 1977)

It is important to consider the specific materials and the characteristics of the fly ash conglomerate. The Kingston Fossil Fly ash make-up includes "trace amounts of metals" (PHA) The availability of metals in the conglomerate is another key factor ignored by Plaintiffs and their experts. Exposure to fly ash conglomerated particles is not same as exposure to the individual metals in the fly ash. The individual metals are bound (conglomerate) within the fly ash particles and the fly ash particles are "stable under most conditions". (*See MSDS)* The health hazard of fly ash particles is equivalent to that of other common dust particles. (PHA) The toxicological properties of coal fly ash indicate the ash can be considered a nuisance dust. This means that the Kingston fly ***ash would have the same potential adverse health effects as other mineral particulates (dust). The metallic content of the fly ash would not make the particulate matter more toxic. Fly ash does not appear to have greater potency to cause pulmonary effects than other ambient particulates.*** [*See Tennessee Department of Health* Public Health Assessment (PHA), Page 43.] Specifically, the Tennessee Department of Health's PHA found:

- "Remember, the metals are bound to the ash." (PHA, Page 20) Thus, before people could be exposed to the metals bound in the ash (as opposed to mechanical exposure to the ash particles themselves), the metals somehow would have to become unbound from the ash as by leaching from the ash particles or by decomposition of the ash particles.

- Laboratory testing of and actual experience with Kingston ash particles shows there is little leaching of the metals bound in Kingston ash particles. Thus, the PHA points out that"[l]aboratory results indicated that ***very little*** of the metals leached from the coal ash." (PHA, Page 16).

- Similarly, the report of the EPA Science Panel Review states that "[t]he surface water monitoring data reported to date demonstrates that most metals and metalloids are ***not*** readily leaching off of the particles . . . ." (EPA Science Panel Review, Page 2).

The lack of leaching is further supported by the biomonitoring tests done on Plaintiff's which found no elevated levels of metals. (See Biomonitoring Table)

ANALYSIS OF CAUSATION: THE IMPORTANCE OF THE DIFFERENTIAL DIAGNOSIS

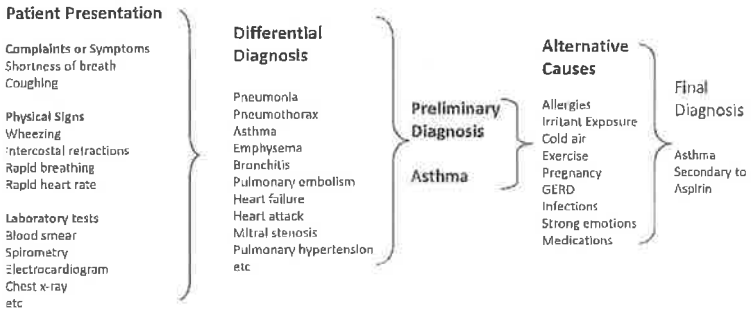
The process of differential diagnosis is distinguishing the most likely disease from a list of possibilities that present with similar clinical characteristics. A medical complaint alone cannot be used to determine dose, exposure or a source. If an individual has contacted a substance (exposure), has received a dose of a sufficient quantity and a recognized medical condition develops, then a differential diagnosis must be performed to consider all possible etiologies known to present with the objective findings. A consideration of alternative causes for a diagnosis must be considered. Then causation analysis must be employed to determine if the illness is the casually connected to the substance. Typically, the causation criteria put forth by Sir Austin Bradford Hill (Strength, Consistency, Specificity, Temporality, Biological Gradient, Plausibility, Coherence, Experimental, and Analogy) are used to determine causation, and are further discussed below (*See infra* page 16; Hill 1965); others also are used. (Doll 1984, Evans 1976, Guidotti 1986, Hackney 1979) These are commonly known as the Hill Criteria and were proposed in 1965 by Sir Austin Bradford Hill in his studies on the relationship between tobacco smoking and lung cancer.

If the individual has been exposed, has received a dose, and the dose is of a sufficient magnitude to cause an injury, then there must be a compatible medical condition. Evaluation of the medical condition is achieved through the traditional approach including a history, a physical examination, and the application of medical laboratory tests. From these, a list of possible diagnoses, formally called differential diagnosis, is assembled. Not to be lightly undertaken, differential diagnosis is nuanced and complex; entire texts are devoted to the differential diagnosis of signs and symptoms. This most likely diagnosis then becomes the subject for a formal toxicological causation analysis. (Letz 2014) See the exposure paradigm figure in the following section.

In diagnostics, a symptom (subjective) is a sensation perceived and/or reported by the individual,

as contrasted with a physical sign (objective) that an examiner may see, feel, or hear. Medical diseases are composed of subjective complaints, which are confirmed by a physical examination. Then laboratory tests, or other testing modalities may be employed to narrow the diagnosis.

Differential Diagnosis Example



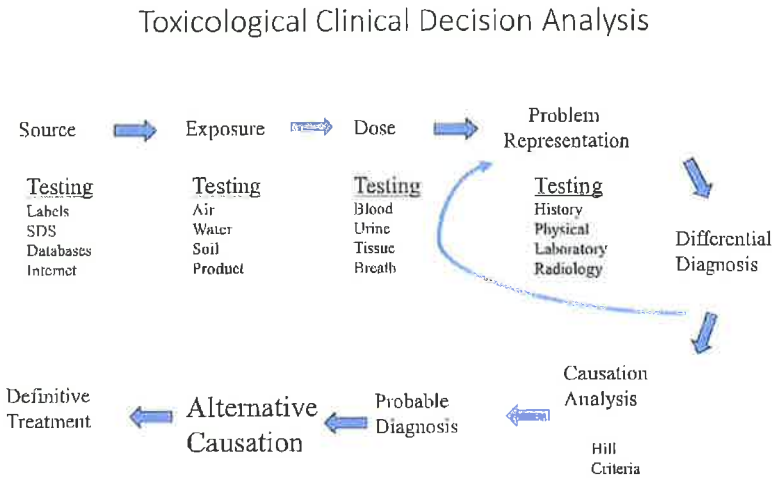
Complaints (symptoms) are subjective, and must be supported with objective (physical signs, testing) data. Medical complaints related to a substance can also be caused by other etiologies. A chemical may produce several different kinds of physiological perturbations to different tissues depending on the dose and duration at which it was been received. Chemicals may produce a range of health effects that overlap with, or may be identical to, those effects caused by infectious agents, genetic deficiencies or other factors based on dose-response. Therefore, all causation criteria should be considered, not simply the specific disease or range of conditions reported in an individual, before arriving at a final opinion.

Plaintiffs' clinical reasoning is fatally flawed. Plaintiffs are reasoning by the *post hoc, ergo propter hoc* (after the fact, therefore, because of the fact) methodology. That is, the alleged symptoms and ailments were used to explain that sufficient exposure had occurred. Then plaintiffs incorrectly assume exposure has been shown to be sufficient, and this "proof of exposure" becomes a basis for explaining the cause of the symptoms and ailments. In short, the subjective claims fundamentally become the basis for explaining themselves. This is fatal circular reasoning. Such circular reasoning is not scientifically or medically acceptable.

Dr. Rea and Dr. Terry have not used accepted methodology in reaching their conclusion that the Plaintiffs have illness due to fly ash exposure from the Kingston site. There is no discussion of exposure or dose, which are fundamental to causation analysis. This is a ruinous flaw in the methodology of Dr.'s Rea and Terry that nullifies their methodology. Additionally, Dr. Ellis, Dr.

Dhand, Ms. Landaiche, Mr. Williams, and Dr. Draughon have also failed to use accepted methodology in reaching their conclusions.

An example of an accepted linear reasoning process follows:



As discussed previously, in order to establish causation, it is mandatory to logically and sequentially establish that (1) a source exists, (2) a complete pathway exists from source to potentially exposed individuals, (3) a concentration of the chemical of interest is either reliably measured or calculated at the exposure point(s), (4) the dose resulting from contact with exposure media is interpreted in a scientific manner based on the known toxicology and published dose-response literature, (5) the observed health effects are reconsidered in light of the known dose-response effects, and (6) an appropriate causation list is constructed. Therefore, the mere presence of a chemical does not necessarily imply that adverse health effects will either occur or be attributable to that chemical. Rather, a conclusion that exposure to a chemical causes a disease in an individual (as opposed individual merely being contemporaneous with chemical) must be based on findings that the occurrence of the disease is significantly increased in the group with chemical exposure in comparison with an otherwise similar non-exposed group, with the weight of evidence confirming that these results are not due to chance, bias, or confounding. Plaintiffs have not demonstrated a statistically significant increase in diseases attributable to fly ash.

The Hill criteria are a series of scientific considerations or attributes to be considered in evaluating disease causation (Hill 1965). The Hill Criteria delineate specific types of data that should be used as foundation for differentiating true causal associations from indirect or artefactual findings: (1) strength of association (what are the mathematical odds of developing the outcome of interest from

being exposed to the proposed agent); (2) consistency of the association (do we see the same outcomes in different studies with different populations and designs); (3) specificity of association (is the association limited to a single cause and effect); (4) temporality (time sequence, i.e., the exposure must precede the health effect) of the association; (5) biological gradient (increasing risk or severity of the outcome of interest in association with an increased dose) observed; (6) biologic plausibility (is there agreement with our current understanding of how cells and organs react and respond); (7) coherence (are the results in agreement with our current understanding of the distributions of causes and outcomes in humans); (8) experimental or intervention effect (do the observed effects decrease or stop when the exposure is removed); and (9) analogy (are there other similar chemicals that are known to act in the same fashion).

These criteria are generally considered to represent a useful methodology or process for evaluating data for evidence of causation. It is not necessary to simultaneously meet and fully satisfy all nine considerations. Conversely, it is inappropriate to selectively choose individual criteria while ignoring obvious information that is clearly applicable to other criteria. For example, the ability to construct a mechanism of action that appears biologically plausible is not alone sufficient for attribution of causation if multiple other categories of data and observations are incompatible with the proposed "plausible" hypothesis. To meet a reasonable standard for causation, the Hill Criteria illustrate that a systematic rather than a selective review of all reliable and relevant data is necessary.

It is important to recognize that even if the weight of evidence supports a general causal relationship between exposure to a chemical and a given human disease, it does not necessarily mean that a specific individual's manifestation of that disease resulted from exposure to the chemical. Attributing a disease in a specific individual to chemical exposure without carefully demonstrating that a complete exposure pathway with a toxicologically significant dose; followed by a well-constructed causation analysis, is inappropriate and leads to circular logic. It is not sufficient to simply argue that (1) chemical "A" is present in the proximate environment to a group of individuals, (2) chemical "A" can cause effect "Y", (3) some people in the group report effect "Y", therefore, (4) chemical "A" is the explanation for the occurrence of effect Y in the group. Such logical fallacies can only be avoided by rigorous application of the systematic methodology described above, which I have employed in this report.

Plaintiffs have failed to follow a generally accepted methodology in reaching their opinions of a hypothetical relationship between fly ash and health effects. Plaintiffs have not provided objective evidence of an elevated exposure nor elevated dose(s) to arsenic or other chemicals of potential concern (based on their own sampling). In fact, the results of the heavy metal testing that has been done on plaintiffs shows that heavy metals were not detected at all or were detected at minimal levels of no medical concern. This disproves Plaintiffs' own assertions that they were exposed to elevated levels. The absence of proof of elevated levels in all of Plaintiffs makes it impossible to establish causation.

Even their own studies prove otherwise. They have provided no evidence of a scientific inverse relationship between the level of fly ash and the level of testosterone (no dose response relationship), meaning that they have provided no evidence that as the level of fly ash increased, the level of plaintiffs' testosterone decreased. In fact, animal studies suggest that with exposure to fly ash, there was either no change in testosterone levels or an *increase* in circulating levels of testosterone. (Hopkins 1997, Ward 2006). Moreover, plaintiffs have provided no evidence of a scientific relationship between fly ash and the other alleged diseases. Plaintiffs' opinions are speculations, without objective evidence of a causal nexus. The plaintiffs' expert reports do not support general causation.

Plaintiffs also claim low testosterone (Low-T) because of fly ash exposure. There are no scientific peer reviewed papers that would support such a claim. There are several causes of secondary hypogonadism with low testosterone. These may include:

Congenital Abnormalities
- Isolated hypogonadotropic hypogonadism
- Other hypothalamic pituitary deficits

Acquired Diseases
- Suppression of gonadotropins
  - Hyperprolactinemia
  - GnRH analogs
  - Gonadal steroids
  - Glucocorticoid treatments
  - Continuous opiate administration
  - Critical Illness
  - Chronic systemic illness
  - Anorexia nervosa
  - Diabetes Mellitus
  - Obstructive Sleep Apnea
- Damage to Gonadotrophic cells
  - Benign tumors and cysts
  - Malignant tumors
  - Infiltrative diseases
  - Infections
  - Pituitary apoplexy
  - Trauma
  - Ischemic
  - Idiopathic

It is important to note that Dr. Rea did not test for or diagnose low testosterone or hypogonadism in the plaintiffs. There is one animal study that suggests increased testosterone from fly ash exposure. (Hopkins 1997)

CRITERIA FOR MEDICAL MONITORING

In the field of toxicology, we often hear of claims or fears of future disease. Some suggest a

remedy for "fear" is a "medical monitoring program". Anyone attempting to demonstrate the appropriateness and necessity of a medical monitoring program must recognize that such an idea potentially is not only ineffective but frankly also harmful. Because it is widely accepted that programs for early detection of disease may be of no benefit or may be harmful, a broad medical consensus around at least three criteria has materialized for how one decides if medical monitoring is necessary and appropriate. First, a critical analysis must be undertaken of the natural histories of the disease conditions alleged to occur because of the toxic exposure and targeted for medical monitoring. Specifically, tests must be available to identify a condition early enough to allow intervention with medical treatment to change its morbidity or mortality. Next, the effectiveness of each test in a proposed medical monitoring program must be justified either by evidence from a controlled clinical trial of the test, or from a quantitative decision analysis, a modeling technique that can simulate the elements of a controlled clinical trial to rigorously define the likely outcome. Finally, the theoretical risk of future diseases must be sufficiently high, so that if there are tests with proven sensitivity and specificity the prevalence of occult disease is high enough to yield an acceptable positive predictive value. Such tests are difficult to develop even for "high risk" medical conditions. For example, the panel of 300 experts convened by the U.S. Public Health Service to study the scientific and medical appropriateness of various proposed screening tests, concluded that based on data from controlled trials of screening, use of annual chest x-rays to screen asymptomatic smokers, a high-risk group for lung cancer, was not recommended (U.S. Preventive Services Task Force).

There are standard definitions for the terms "medical monitoring," "screening," and "surveillance". Medical screening and medical surveillance are terms often inappropriately interchanged. Medical screening is a clinical strategy focusing on early diagnosis and treatment of an individual. In contrast, medical surveillance is a preventive strategy focusing on the detection and elimination of the underlying causes, i.e., hazards and exposures, of any discovered trends in a worker population. Thus, screening is a method for detecting disease or body dysfunction before an individual normally would seek medical care.

Medical screening is a scientifically defined process that is well established in the medical literature. The scientific criteria for evaluating laboratory procedures that could be used in a medical screening program have been published by several authoritative bodies, including the ATSDR (1995), the U.S. Preventive Services Task Force (USPSTF) (1996), and the WHO (1971). In the United States, ATSDR is the organization charged with delineating scientific criteria for community-based medical screening of asymptomatic individuals exposed to potentially harmful levels of hazardous chemicals. ATSDR (1995) has published formal scientific criteria for determining if medical monitoring is appropriate in a community exposure setting. These criteria are consistent with those previously published by both WHO and USPSTF.

Medical monitoring excludes those with symptomatic disease. Therefore, the focus of the program is not to detect clinically active disease, but rather, medical monitoring focuses on the risk of latent disease resulting from prior exposure. In designing a medical screening protocol, it is important to

consider the work of USPSTF, which established an age-, sex-, and lifestyle-dependent schedule of medical screening tests. Therefore, it would be unnecessary and inappropriate to recommend testing.

Therefore, based on the established published criteria, there is no scientific basis for either a medical screening or medical monitoring program for any of the plaintiffs because the exposures were low, the biomonitoring was low, yet plaintiffs are claiming illness related to the site clean-up. Remediation workers at the Kingston site do not need additional medical monitoring or surveillance beyond that recommended for any individual based on age, sex, other standard lifestyle-dependent behaviors, OSHA, or other pre-existing medical conditions or considerations.

MEDICAL AND TOXICOLOGICAL EVALUATION OF PLAINTIFFS

General Causation Analysis for Plaintiffs

It is generally accepted within the medical and scientific communities that evidence of general causation requires positive epidemiological studies that establish an association between exposure to a chemical and specific adverse health effects (Section 7.1). Dr. Rea, Dr. Terry, Dr. Ellis, Dr. Dhand, Ms. Landaiche, Mr. Williams, and Dr. Draughon have presented no general or specific causation analysis, simply attributing the claimed medical illnesses to exposures to fly ash. Clearly, because of these failures, Dr's. Rea, Terry, Dhand and Draughon's opinions regarding causation are completely unfounded, unscientific and fail to follow accepted medical or toxicological methodology.

Plaintiffs' Diseases

Vital to conducting an accepted medical toxicological investigation, the expert must identify the source, exposure, dose, and medical condition of the affected individual(s). The source of the chemical substance at issue must be identified, there must an opportunity for physical contact with the chemical at issue, the amount of the chemical to which the individual is exposed must penetrate inside the human body, and if exposure occurs and a dose is received, then the dose must be of sufficient magnitude to cause a medical condition documented to be associated with the alleged exposure. A formal causation analysis is then applied to the objective data in the case, and a decision determined on a weight of the evidence basis.

Lacking any evidence of general causation for these plaintiffs, the concept of specific causation is irrelevant.

Specific Causation Analysis for Plaintiffs

In the absence of credible evidence for general causation, there is no basis for analysis of specific causation. A specific causation analysis considers:

- Whether the specific plaintiff was exposed to a sufficient concentration to the released

substance, and the duration;[3]

- The availability of objective exposure data available for the specific residence under consideration;
- The bioavailability (particle dissolution) of various metals from the conglomerate with elevated heavy metals in an individual;
- The measured chemical concentration data;
- The relationship between the objectively measured exposure data and the disease endpoint under consideration, i.e., the dose-response relationship;
- Alternative causation analysis, including relevant risk factors such as age, gender, lifestyles, pre-existing illnesses, and treatments (medications, radiation or chemotherapy, etc.).

Because general causation has not been met, I have NOT considered, from a medical toxicology perspective, the issue of specific causation for plaintiffs. I reserve the right to opine on the specific cause evidence plaintiff's claim support their allegations. It is my opinion health effects were not produced because of fly ash exposures during the cleanup of the TVA Kingston Fossil release.

Plaintiffs' experts have provided no quantification of the dose of the fly ash received by plaintiffs, and thus plaintiffs' experts cannot know if the alleged dose was of sufficient magnitude to cause a medical condition. Plaintiffs' experts' dose speculation is not generally accepted in the medical community in establishing a pivotal nexus between exposure and disease.

Plaintiffs allege that their diseases were caused by exposure to fly ash and other substances associated with an earthen dike breach from the TVA Kingston Fossil power plant facility site with an ensuing release on 12-22-2008. Health claims by plaintiffs are listed in the following table.

List of Plaintiffs' Specific Claims

Name	Age	Claimed Signs and Symptoms
Greg Adkisson	47	Black spots in both lungs, SOB
William Bass	68	Wheezing, breathing problems, hearing loss
Gabriel Billingsley	41	Fatigue, ED, Low T, skin lesions, sinus problems, HTN
Leonard Bledsoe	73	Heart, breathing and sinus problems, Low T, rash, Stroke (2009)
Carl Booker	66	Stroke, lung infection, skin cancer, emotional distress

[3] Dr. Rea's laboratory tests did not demonstrate elevated metals in the plaintiffs. Though Dr. Rea claims the plaintiffs are sick form metals, his own tests do not support his opinion.

Jeffrey Brewer	42	Low T, breathing and sinus problems, blood pressure, fatigue, CP, lung disease, eyes burning, HA, rash
Johnny Church	67	Chronic Lymphocytic Leukemia (2009), blood pressure
Ansol Clark	66	Atrial fibrillation, allergies, CHF, stroke, Polycythemia vera, Low T, enlarged prostate, eye damage due to stroke
Danny Cody	58	HTN, high blood sugar, cough, rash, Low T, heart and breathing problems
John David Cox	56	Breathing and skin problems, heart arrhythmia
Phillip Crisk	59	Severe sinus problems, HA, rapid heartbeat, HTN, T2DM (since 42), extreme fatigue, blisters and skin rash
Joe Cunningham	29	No specific allegations of illness or injury secondary to workplace toxic exposure were included in the medicals or accompanying documentation.
Enoch Edmonds	71	COPD, heart problems, rectal cancer (2013)
Billy Gibson	44	Asthma, Low T, sinus problems
William Hedgecoth	58	Circulatory problems, blockage of blood flow to lower extremities, back, neck and spine pain, arthritis, HTN
Stanley Hill	53	Heart, breathing and sinus problems, low T, skin lesions, HA, memory loss
William Isley	41	Sinus problems, HA, HTN, Low T, skin lesions, arsenic poisoning in blood
David Jones	48	SOB, nose bleeds, skin cancer, Low T, pain in arms and legs, numbness in legs, sinus infections
Jimmy Kilby	49	Lungs, sinus, quadruple bypass, heart disease, GI problems, surgery, skin problems, skin lumps, aggravated diabetes
Clint Mannis	52	Chronic sinus infections, respiratory infections, skin lesions, low vitamin D, kidney problems/stone, eye lesion, neurological problems-neuropathy in feet
Michael MacCarthy	54	Eye, sinus, pulmonary, heart, and other health-related problems, neurological, pulmonary, prostate, heart and sinus problems
Frankie Norris	56	HTN, enlarged heart, loss of memory, sinus problems, restless leg syndrome, weight loss, gout, nerve pain in legs, watery eyes, mood swings, excessive bruising, soreness in arms
Nicholas E Perry	33	Pulmonary and sinus problems, Low T
Joseph Randall Pursiful	58	Breathing, emotional distress, Low T, upper respiratory, sinus surgery, rash on skin, conflict with spouse, repeated bronchitis
Ralph G. Ramey	37	Injury to his heart, and was sick-behind on jobs
Bill Rose (Deceased)	59	AML, rectal cancer, dysphagia
Michael Shelton	53	No demand letter was submitted with the medicals so the specific allegations are unknown. Diagnosed and treated for an MI (20014) and lung cancer (2015). He subsequently

		died secondary to complications from the surgery to address the lung cancer.
Shaun Smith	38	Skin cancer (Squamous cell), nodular prurigo disease, sinus problems, pulmonary, constant headaches, delayed wound healing due to prurigo
Brian Summers	39	HA, nose bleeds, prostate problems, bronchitis, sinus problems, pain in testicles, rash, memory loss, problems staying awake, high arsenic level
Brian Thacker	47	Blisters, chronic bronchitis, headaches, sinus problems, Low T, depression
Kevin Thompson	37	Fluid on lungs, spots on lungs, sinus trouble, nose bleeds, severe allergic reactions, asthma,
Craig Wilkinson	59	COPD, on lung transplant list
Donald Van Gilder	42	Fear from Disease , Leukemia

Analysis of Plaintiffs' Exposures

Comprehensive and extensive air sampling was done for:

Total Dust
Respirable Dust
Silica
Aluminum Oxide
Aluminum
Antimony
Arsenic
Barium
Beryllium
Cadmium
Calcium Oxide
Chromium Metal
Cobalt
Copper
Iron Oxide
Quartz
Lead
Lithium
Magnesium
Manganese
Molybdenum
Nickel
Potassium
Selenium
Sodium
Thallium
Vanadium
Zinc Oxide

Air sample concentrations were very low for all metals and materials tested and were below both the OSHA levels and site safety levels. The measured exposure was insufficient to cause adverse health effects.

Samples also were compared between job descriptions (dozer and excavator operators, flaggers, scraper pan operators, artic dump and water truck operators). Air sample concentrations were very low, with rare exceptions. Flaggers, who are not inside a closed vehicle cab, had average total dust measures of 0.0428 mg/m^3 (OSHA-PEL- 5 mg/m^3). (CP SEG Summary)

Critique of Plaintiffs' Experts' Exposure Characterization

Key to a reliable exposure assessment are the Kingston Fossil power plant remediation area air measurements. Plaintiffs' experts have not opined on the measures. Fundamental to an exposure assessment is the identification of the quantification of the plaintiffs' dose, the movement of the substances though the body (toxicokinetics) and the potential interaction with various organs or body systems ("dose-response"), both in terms of how the chemical is initially distributed through the organism as well as how it ultimately produces a specific ill-effect (toxicodynamics). Plaintiffs' experts provided no exposure assessment. Plaintiffs have not provided their causation methodology or followed a generally accepted causation methodology for toxicology. (AMA 2013)

On behalf of plaintiffs, Dr. Rea has been endorsed to testify that their exposure to fly ash caused adverse health effects. In the Disclosure of Dr. Rea, he offers the following opinions:

> "Dr. Rea will testify that fly ash constituents are toxic and, in certain instances, carcinogenic. He will also testify that when chronic exposure occurs, fly ash causes a variety of health problems that are and/or were the medical cause of death, health problems, disability, loss of enjoyment of life, pain, suffering, loss of function and aggravation of a host of different medical conditions of the Plaintiffs including, but not limited to: leukemia, polycythemia vera (PCV), other blood disorders, lung cancers, skin cancers, intestinal cancers, lower rectal cancers, pulmonary disease, heart disease, stroke, various neurological conditions, various skin conditions, diabetes, sinus problems, low testosterone, oxygenation problems, and an overall injury and/or damage to the Plaintiffs' immune function." (Disclosure of William Rea, MD)

There are limited data in high dose animal experiments of lung inflammation with direct instillation of fly ash into the trachea of rodents. There is no data in humans that would support a general causation nexus between fly ash and the above outcomes endorsed by Dr. Rea. Dr. Rea provides no discussion of the air concentrations, biological monitoring or a dose response relationship. Dr. Rea's opinions are fatally flawed, and unsupported in the scientific literature. Chemicals cause specific physiological changes and medical effects. In medicine, we select specific chemicals based on target organ specificity, mechanism of action and a favorable risk benefit. We call these medications. Medications (which are chemicals) have very specific effects (toxicodynamics). For Dr. Rea to imply the long laundry list of effects is causally related to fly ash in his endorsement is scientifically baseless. Furthermore, Dr. Rea's approach is a reckless disregard for proper scientific methodology. There is no evidence for general causation in this case.

Dr. Rea is a chest surgeon and alternative care practitioner. Dr. Rea is not a medical toxicologist, and to my understanding has no training or specific certification in toxicology by any recognized toxicology board.

Furthermore, Dr. Terry, Mr. Ellis, Dr. Dhand, Ms. Landaiche, Mr. Williams, and Dr. Draughon (like Dr. Rea) have personally not measured air concentrations or biological samples from plaintiffs as a means of characterizing and quantifying the alleged exposure.

ENCEPHALOPATHY

Encephalopathy is a global (diffuse) brain insult that causes an alteration in a person's mental status. The quintessential encephalopathic substance is alcohol. Acute alcohol intoxication, and chronic brain injury from alcohol are well known and commonly seen. Many chemicals including medications may cause encephalopathy and the list of causes is extensive. The National Institute of Neurological Disorders and Stroke (NINDS) has described encephalopathy as:

> "Encephalopathy is a term for any diffuse disease of the brain that alters brain function or structure. Encephalopathy may be caused by infectious agent (bacteria, virus, or prion), metabolic or mitochondrial dysfunction, brain tumor or increased pressure in the skull, prolonged exposure to toxic elements (including solvents, drugs, radiation, paints, industrial chemicals, and certain metals), chronic progressive trauma, poor nutrition, or lack of oxygen or blood flow to the brain. The hallmark of encephalopathy is an altered mental state. Depending on the type and severity of encephalopathy, common neurological symptoms are progressive loss of memory and cognitive ability, subtle personality changes, inability to concentrate, lethargy, and progressive loss of consciousness. Other neurological symptoms may include myoclonus (involuntary twitching of a muscle or group of muscles), nystagmus (rapid, involuntary eye movement), tremor, muscle atrophy and weakness, dementia, seizures, and loss of ability to swallow or speak. Blood tests, spinal fluid examination, imaging studies, electroencephalograms, and similar diagnostic studies may be used to differentiate the various causes of encephalopathy."[4]

Chronic encephalopathies are portrayed by a chronic mental status change that is slowly progressive. An exception to this is anoxic encephalopathy (brain injury from lack of oxygen). Chronic causes are usually irreversible, structural changes in the brain. If discovered early, some causes may be halted or reversed. Examples include: low oxygen, trauma, chemicals such as alcohol; HIV; some chemotherapy; hereditary enzyme deficiencies; and Korsakoff (from Wernicke syndrome).

Acute encephalopathy is characterized by global, functional alteration of mental status due to systemic factors. It is typically reversible when these abnormalities are corrected, with a return to baseline mental status. Acute encephalopathy may be further identified as toxic, metabolic, or toxic-metabolic. Toxic encephalopathy describes acute mental status alteration due to medications,

[4] https://www.ninds.nih.gov/Disorders/All-Disorders/Encephalopathy-Information-Page

illicit drugs, or other chemicals. Metabolic encephalopathy is caused by many metabolic disturbances. Toxic-metabolic encephalopathy describes a combination of toxic and metabolic factors. Causes of toxic-metabolic encephalopathy includes organ failure such as hepatic and renal; alcohol (the quintessential encephalopathic agent, both acute and chronically); dehydration; electrolyte imbalance; fever; hypertension; hypoxemia; illicit drugs; infections including sepsis; medications; toxic chemicals; and Wernicke (thiamine deficiency).

Evaluation of Potential Alternative Causes of Plaintiffs' Diseases

In their disclosures, plaintiffs' experts did not reveal the methodology they used to eliminate confounders, or alternate causes for the plaintiffs' complaints. A few possibilities for alternative causes include: other exposures, lifestyle, hobbies, inheritable factors and spontaneous causes. Therefore, since plaintiffs' experts have failed to reveal the methodology they used to eliminate alternate causes for the plaintiffs' complaints, their reports fail to show the alleged chemical causation can definitively be ruled in.

Plaintiffs do not allege "Black Lung Disease" also known as coal workers' pneumoconiosis. Plaintiffs' complaints are distinguishable from coal workers' pneumoconiosis. Pneumoconioses can be subdivided into fibrogenic (eg, silica, coal, talc, asbestos), benign or inert (e.g., iron, tin, barium), granulomatous (e.g., beryllium), and giant cell pneumonia associated with hard metal inhalation (e.g., cobalt). Coal worker's pneumoconiosis (CWP) may results from inhalation and deposition of silica-free coal dust particles that induce the formation of coal macules, once they reach the air sacs of the lungs. CWP has similar radiographic features to silicosis, but is classified as a separate disease due to its rather characteristic pathologic findings. It is not uncommon to have a mixed picture that also includes silica dust exposure. In simple forms, CWP patients accumulate pigment and reticulin fibers in a peribronchiolar location. Over time, large amorphous black masses develop that occasionally have a liquefied center. Subsequent cavitation may occur. The International Labor Organization (ILO) classification emphasizes the combination of radiologic suspicion and exposure history.

There is no evidence the plaintiffs are suffering from silicosis or coal workers' pneumoconiosis related to the Kingston site. Dr. Rea has not provided the requisite diagnostic support for this allegation.

DISCUSSION OF OPINIONS

Opinion 1: Plaintiffs' experts did not use accepted scientific methodology in reaching their conclusions regarding plaintiffs' exposures and risks due to exposure to fly ash. Plaintiffs' claims do not fulfill general causation.

The generally accepted scientific methodology for exposure and health risk assessment requires that chemicals of potential concern (COPCs) be clearly identified as (1) arising from the source under study, (2) present at concentrations significantly greater than background in the plaintiffs' homes, and (3) causally related to disease endpoints of concern under documented exposure conditions. Plaintiffs' experts have failed to characterize exposure and dose, essential components of the Source → Exposure → Dose → Potential Health Effect(s) paradigm. The plaintiffs do not

meet the weight of the evidence approach using the Hill criteria, and therefore general causation has not been met. Plaintiffs have no strength of association (plaintiffs have provided no epidemiological studies supporting fly ash and claimed health conditions), specificity (plaintiffs have not established fly ash causing specific health effects in plaintiffs), consistency (variance in health claims by plaintiffs), temporality (associated weakly through employment, but lacking confidence without considering pre-employment medical records), biological gradient (no dose or dose-response relationship established in plaintiffs), plausibility has not been met for the claimed illnesses, coherence (claims are not consistent with dose-response and literature), experimental (plaintiffs have provided no experimental evidence of a causal nexus), or analogy (plaintiffs have not provided an analogous exposure in other species or individuals).

Opinion 2: There is no medical basis for fear of developing diseases because there is no evidence that any plaintiff was exposed to levels of fly ash or any other released chemicals that could cause any long-term injury.

Measured air levels of total dust were noted to be very low. Fly ash at these low concentrations do not cause adverse health effects. The mean respirable dust concentrations for all job descriptions is 0.044 mg/m^3, with an OSHA PEL of 5 mg/m^3. This is based on the Jacobs IH job category analysis. (CP SEG Summary document)

Numerous studies have examined the potential health effects of fly ash and have found at most limited transient effects. (Fisher 1983, Liberda 2013, MacFarland 1970, Raabe 1982) Liberda (2013) studied fugitive dust emissions at the Kingston Fossil Plant, and found that the doses of dust were insufficient to cause adverse health effects in humans. It is my opinion as a medical toxicologist that no health effects have, or will, occur in relation to the Kingston Fossil Plant clean up.

Opinion 3: There is no evidence that plaintiffs were exposed to harmful levels of fly ash or any other chemicals during the Kingston Fossil Plant clean up.

Measured air levels of total dust were noted to be very low. Fly ash at these low concentrations do not cause adverse health effects. The mean respirable dust concentrations for all job descriptions is 0.044 mg/m^3, with an OSHA PEL of 5 mg/m^3. This is based on the Jacobs IH job category analysis. (CP SEG Summary document)

Opinion 4: In this case, there is no scientific basis for recommending medical monitoring.

As discussed previously, there are generally accepted criteria for determining if individuals in a potentially affected community should be provided medical monitoring. These criteria include documented evidence of exposure to a hazardous substance, which did not occur in this case. There were no elevated air or arsenic levels. If there is evidence of such an exposure, each person is evaluated individually. In this case, none of the criteria have been met. Accordingly, there is no scientific basis for recommending medical monitoring for plaintiffs.

The plaintiffs' alleged complaints illustrate a wide variety of medical conditions that do not demonstrate a pattern of diseases attributable to exposure to fly ash or other spill chemicals. Rather, the plaintiffs' health conditions most likely are associated with specific lifestyle habits and choices and genetic makeup.

In summary, the monitoring data of exposure information in this case indicate low levels of dust that are not associated with acute or chronic health effects. Plaintiffs' complaints are significantly different from one another in multiple variables relevant to exposure and susceptibility. From a toxicology perspective, plaintiffs have not met the weight of the evidence (general causation). There are no anticipated long term effects based on exposure measures, and medical monitoring is not indicated.

I charge $425.00 per hour for all work performed on this case. A copy of my CV and list of testimonies is available.

Scott D. Phillips, MD

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CURRICULUM VITAE

Scott D. Phillips, M.D., F.A.C.P., F.A.C.M.T., F.A.A.C.T.

Fellow of the American College of Physicians
Fellow of the American College of Medical Toxicology
Fellow of the American Academy of Clinical Toxicology

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PO Box 13250
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ACADEMIC APPOINTMENTS

Associate Clinical Professor
Division of Clinical Pharmacology & Toxicology, Department of Medicine, University of Colorado Health Sciences Center, Denver, CO, 1999 - Present

Associate Clinical Professor
Division of Emergency Medicine, Department of Surgery, University of Colorado Health Sciences Center, Denver, CO, 2002 - 2004

Assistant Clinical Professor
Occupational Health Services Division of Emergency Medicine, Department of Surgery, University of Colorado Health Sciences Center, Denver, CO, 1993 - 1999

Assistant Clinical Professor
Division of Clinical Pharmacology & Toxicology, Department of Medicine, University of Colorado Health Sciences Center, Denver, CO, 1994 - 1999

Clinical Instructor
Division of Emergency Medicine, Department of Surgery, University of Colorado Health Sciences Center, Denver, CO, 1992 - 1993

Clinical Instructor

Division of Clinical Pharmacology & Toxicology, Department of Medicine, University of Colorado Health Sciences Center, Denver, CO, 1992 - 1993

Assistant Director
Occupational Health/Environmental Toxicology Clinic, University of Colorado Health Sciences Center, Denver, CO, 1994

PROFESSIONAL AFFILIATIONS

Partner, NewFields, LLC, Denver, CO

PROFESSIONAL APPOINTMENTS

Attending Physician
Department of Medicine, Medical Toxicology, Sky Ridge Medical Center, Lone Tree, CO, 2005 - 2010

Attending Physician
Division of Clinical Pharmacology & Toxicology, Department of Medicine, University Hospital, University of Colorado Health Sciences Center, Denver, CO, 1992 - Present

Attending Physician
Denver Health Medical Center, Department of Medicine/Toxicology, Division of Medical Toxicology, Denver, CO, 1992 - Present

Attending Physician
The Children's Hospital, Department of General Pediatrics, Toxicology Privileges, Denver, CO, 2002 - Present

Attending Physician
Porter Adventist Hospital Regional Poison Treatment Center (Adult and Pediatric Privileges), Denver, CO 1990 - 2015

Attending Physician
Littleton Adventist Hospital, Toxicology, Littleton, CO, 1993 - 2015

Attending Physician
Swedish-Columbia Medical Center Internal Medicine & Toxicology, Englewood, CO, 1990 - 2014

Attending Physician
Rocky Mountain Poison and Drug Center and Clinical Toxicology Service, Denver, CO, 1993 – Present

Attending Physician
Washington Poison Center 2016- Present

Attending Physician
Rocky Mountain Poison Center Medical Toxicology Fellowship Task Force, Denver, CO, 1995 - Present

Attending Physician
Baromedical Physicians at PorterCare Hospital
Regional Baromedical Center, Denver, CO 1993 - 2000

Attending Physician
Occupational Medicine & Toxicology, Coors' Industries Medical Center, Golden, CO, 1992 - 1996

Attending Physician
Emergency Department, Landmark Medical Center Woonsocket, Rhode Island, 1988 - 1990

EDUCATION

College:
Gonzaga University, Spokane, WA 1974 - 1978
Degree: B.S. Biology

Graduate School:
Washington State University, Pullman, WA 1978 - 1980

Medical School:
American University of the Caribbean School of Medicine, Montserrat, British West Indies 1980 - 1984, Degree: M.D. January 1984

Graduate Medical Education:
Internship: Internal Medicine, Framingham Union Hospital/Boston University, School of Medicine, Framingham, MA. Graduated June 1984 - 1985

Residency: Internal Medicine, Framingham Union Hospital/Boston University, School of Medicine, Framingham, MA. Graduated June 1985 - 1987

Chief Medical Resident: Internal Medicine, Framingham Union Hospital/Boston University, School of Medicine, Framingham, MA. June 1987 - 1988

Fellowship: Clinical Toxicology, Rocky Mountain Poison and Drug Center, Denver General Hospital, University of Colorado Health Sciences Center. 1990 - 1992

COURSES

Foundations of Doctoring Physical Examination/Communication 2005-Present
University of Colorado Health Sciences Center, Denver CO

PRMD 6615 Topics in Environmental & Occupational Health 2006
Preventive Medicine and Biometrics
University of Colorado Health Sciences Center, Denver CO

PRMD 6615 Topics in Environmental & Occupational Health 2007
Preventive Medicine and Biometrics
University of Colorado Health Sciences Center, Denver CO

BOARD CERTIFICATION

Diplomate – Joint American Boards of Preventive Medicine, Pediatrics and Emergency Medicine, Sub-Specialty Sub-Board on Medical Toxicology, Re-Certified 2016 - 2026

Diplomate – Joint American Boards of Preventive Medicine, Pediatrics and Emergency Medicine, Sub-Specialty Sub-Board on Medical Toxicology, Re-Certified 2005 - 2015

Diplomate – Joint American Boards of Preventive Medicine, Pediatrics and Emergency Medicine, Sub-Specialty Sub-Board on Medical Toxicology, Certified 1995-2005

Diplomate – American Board of Internal Medicine, Certified 1987 (Permanent Certificate No. 114277)

LICENSURE

Washington	# 60546792 (Active)
Colorado:	#30404 (Active)
Rhode Island:	#7092 (Inactive)
Pennsylvania:	#MD-037713-E (Inactive)

AWARDS/HONORS

University of Colorado Denver - Outstanding Clinical Faculty: International Service Award 2009-2010

Laureate Award, Colorado Chapter, American College of Physicians, 2009

Outstanding Volunteer Faculty Award. Denver Health Medical Center, 2007

Department of Medicine's Physical Exam Instructor Award for the Foundation of Doctoring Program, University of Colorado, 2007

Top Consultant, Annals of Emergency Medicine, 2001

University of Colorado Health Sciences Center - Outstanding Clinical Faculty Community Service Award 2001

Top Consultant, Annals of Emergency Medicine, 2000

Fellow of the American College of Medical Toxicologists, 1999

Top Consultant, Annals of Emergency Medicine, 1999

National Environmental Health Association, Certificate of Appreciation, 1999

National Environmental Health Association, Certificate of Appreciation, 1999

University of Colorado Health Sciences Center - Outstanding Clinical Faculty Academic Publications Award 1998

National Environmental Health Association, Certificate of Appreciation, 1998

American Association of Poison Control Centers and Micromedex, Inc. – Best Scientific Paper Award 1997

American College of Physicians, Preceptorship Award, 1997

National Environmental Health Association, Certificate of Appreciation, 1997

American Academy of Family Physicians, Certification of Recognition for Teaching, 1996

National Environmental Health Association, Certificate of Appreciation, 1996

National Environmental Health Association, Certificate of Appreciation, 1995

American Academy of Family Physicians, Certification of Recognition for Teaching, 1995

Metropolitan State College of Denver, Recognition for Teaching, 1992

Fellow of the American College of Physicians, 1992

CERTIFICATIONS

Board Certified in Medical Toxicology

Joint American Boards of Preventative Medicine, Pediatrics and Emergency Medicine subspecialty sub board	
Diplomate Medical Toxicology	1995
Re-Certified	2005
Re-Certified	2016

Board Certified in Internal Medicine

Diplomate American Board of Internal Medicine	1987

Colorado Department of Labor and Employment

Workers' Compensation Certification Level II Accreditation, Dept. of Labor Div. or Workers' Compensation	1992
Re-Certified	1996
Re-Certified	2001
Re-Certified	2004

Carolina Hyperbarics

Hyperbaric Medicine	1993

Medical Review Officer Certification Council

Medical Review Officer	1993

Medical Review Officer Certification Council

Medical Review Officer	1998

Pediatric Advanced Life Support 1989

Advanced Trauma Life Support	1987
Advanced Cardiac Life Support	2007-Present
Federation of State Medical Boards	1984
Educational Commission for Foreign Medical Graduates	1983

AMERICAN COLLEGE OF MEDICAL TOXICOLOGY

Medical Toxicology Consultant	Region 8 ACMT-ATSDR Cooperative Partnership
Co-Chair	Liaison Committee (former)
Chair	Occupational & Environmental Sub-Committee of Practice Management Committee (former)
Member	Practice Management Committee (former)
Member	Occupational Environmental Medicine committee (former)
Member	ATSDR Web Based Learning Project (former)

AMERICAN ACADEMY OF CLINICAL TOXICOLOGY

Member	Board of Trustee's	2002-2004
Chair	Communication and Technology Committee	1999 - 2003
Member	Membership Committee (former)	
Member	Research Committee (former)	
Member	Regional Toxicology Treatment Center committee (former)	
Member	*Ad Hoc* Committee on Internet Based Learning (former)	

ADVANCED HAZARDOUS MATERIALS LIFE SUPPORT (AHLS)

Regional Director	AHLS	1999-2001

National Faculty	AHLS (former)	
Member	Science Advisory Committee (former)	

AMERICAN COLLEGE OF OCCUPATIONAL & ENVIRONMENTAL MEDICINE

Chair	Occupational & Clinical Toxicology Committee	1999 - 2005
Member	Council of Scientific Affairs	1999 - 2005
Member	Occupational & Clinical Toxicology Committee (former)	

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION

Member	Emergency Response Planning Guidelines Committee (former)

MEDICAL REVIEW OFFICERS CERTIFICATION COUNCIL

Member	Board of Directors	1996 - 2002
Chair	Nominating Committee of Board of Directors	1999 - 2002

RHODE ISLAND ADVERSE DRUG REACTION REPORTING COMMITTEE

Member		1989 - 1990

HOSPITAL COMMITTEES

Swedish Medical Center

Member	Pharmacy & Therapeutics Committee	2003 - present

Porter/Littleton Adventist Hospital

Advisor	Centura Hospital Research Center	2000 - 2001
Chairman	Human Research IRB, Porter-Littleton Joint Institutional	1999 - 10/2001
Member	Human Research IRB, Porter-Littleton Joint Institutional Review Board	1997 - 2002
Member	Pharmacy and Therapeutics Committee, Porter Adventist Hospital,	1997 - 2002

University Hospital

Member	Safety Committee	1994
Member	Laser Safety Committee	1994
Member	Infectious Disease Committee	1994
Director	Student Health Advisory Committee	1994
Member	Practice Directors Committee	1994

Landmark Medical Center

Member of the Pharmacy and Therapeutics Committee, Woonsocket, Rhode Island, 1989-1990
Medical Director; Woonsocket Fire Department Rescue, 1989 - 1990

Framingham Union Hospital (MetroWest Medial Center)

Member; Coronary Care Committee, 1987 - 1988
Member; Pharmacy and Therapeutics Committee, 1984 - 1985

PROFESSIONAL ORGANIZATIONS

American College of Physicians
American College of Medical Toxicology
American Academy of Clinical Toxicology
American Medical Association
Colorado Medical Society
Denver Medical Society

GRANTS

1. A Multicenter Study of the Efficacy of 4-Methylpyrazole in the Treatment of Methanol and Ethylene Glycol Poisoning.
   - Source: Orphan Medical, Inc.
     Awarded to: Toxicology Associates (Jeffrey Brent, M.D., Ph.D. Principal Investigator, Scott D. Phillips, MD, Kenneth Kulig, MD Co-investigators)
   - Period: 3/1/95 to 12/31/97

2. A Prospective Multicenter Study of Antidepressant Drug Overdoses.
   - Source: Eli Lilly Company
   - Awarded to: The Rocky Mountain Poison Center (Kenneth Kulig, M.D., Jeffrey Brent, M.D., Ph.D., and Scott Phillips, M.D.)
   - Period: 11/1/90 to 11/1/92

3. Professional and Community Education of Vasquez Boulevard – Interstate 70 Site.
   - Source: Association of Occupational Environmental Clinics
     Agency for Toxic Substances Disease Registry

Awarded to: Toxicology Associates, Prof. LLC AOEC Clinic
Period: 4-20-01 to 4-20-02

4. Worker Focused Bio terrorism Education Module.
Source: Association of Occupational Environmental Clinics
Agency for Toxic Substances Disease Registry
Awarded to: Toxicology Associates, Prof. LLC AOEC Clinic
Period: 5-01-02 to 5-01-03

5. Blue Ribbon Panel on Molds in the Building Industry.
Source: North American Home Builders Association
Awarded to: Scott Phillips, MD Panel Chair
Period: 6-1-02 to 12-31-02

6. Conduct peer review and develop educational packages for Case Studies in Environmental Medicine for Chlordane.
Source: Association of Occupational Environmental Clinics
Agency for Toxic Substances and Disease Registry
Awarded to: Scott Phillips, MD
Period: 6-15-05 to 9-30-05

7. Conduct peer review and develop educational packages for Case Studies in Environmental Medicine for Dioxins.
Source: Association of Occupational Environmental Clinics
Agency for Toxic Substances and Disease Registry
Awarded to: Scott Phillips, MD
Period: 6-15-05 to 9-30-05

8. Conduct peer review and develop educational packages for Case Studies in Environmental Medicine for Ethylene/Propylene Glycol.
Source: Association of Occupational Environmental Clinics
Agency for Toxic Substances and Disease Registry
Awarded to: Scott Phillips, MD
Period: 6-15-05 to 9-30-05

9. Conduct peer review and develop educational packages for Case Studies in Environmental Medicine for Nitrate/Nitrite.
Source: Association of Occupational Environmental Clinics
Agency for Toxic Substances and Disease Registry
Awarded to: Scott Phillips, MD
Period: 6-15-05 to 9-30-05

10. Conduct peer review and develop educational packages for Case Studies in Environmental Medicine for Polycyclic Aromatic Hydrocarbons.
Source: Association of Occupational Environmental Clinics
Agency for Toxic Substances and Disease Registry
Awarded to: Scott Phillips, MD

Period: 6-15-05 to 9-30-05

11. Conduct peer review and develop educational packages for Case Studies in Environmental Medicine for Environmental Triggers of Asthma and Ionizing Radiation.
    Source: Association of Occupational Environmental Clinics
    Agency for Toxic Substances and Disease Registry
    Awarded to: Scott Phillips, MD
    Period: 6-15-05 to 9-30-05

12. Conduct pilot testing for CSEMs: Lead, Mercury and Arsenic.
    Source: Association of Occupational Environmental Clinics
    Agency for Toxic Substances and Disease Registry
    Awarded to: Scott Phillips, MD
    Period: 6-15-05 to 9-30-05

DATA SAFETY AND MONITORING REVIEW BOARD

Chair – DSMB 2012-2014

Protocol Title: A Phase III, Multi-Center Clinical Trial of Analatro® [Antivenin Latrodectus (Black Widow) Equine Immune $F(ab)_2$], in Patients with Systemic Latrodectism

Protocol Number: XF-07/03 (version 04) Sponsor of Protocol: Instituto Bioclon S.A. de C.V. in partnership with Rare Disease Therapeutics

GOVERNMENT SCIENCE REVIEW BOARDS

1999 Nominated by the American College of Occupational and Environmental Medicine to the United States Environmental Protection Agency Food Quality Protection Act (FQPA) Science Review Board (SAB) for the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) Scientific Advisory Panel (SAP).

2001 Nominated by the American College of Occupational and Environmental Medicine to the United States Environmental Protection Agency Food Quality Protection Act (FQPA) Science Review Board (SAB) for the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) Scientific Advisory Panel (SAP).

2002 United States Department of Labor, Occupational Safety & Health Administration. Emergency Preparedness and Response. Subject Technical Editorial Board.

JOURNAL EDITORIAL ACTIVITIES

Editor-in-Chief	Internet Journal of Medical Toxicology	2004
Editor-in-Chief	Journal of Medical Toxicology	2005

EDITORIAL ACTIVITIES NON-TEXTBOOK PEER REVIEW

Journal of Medical Risk

CRC Press
Amherst Scientific Publishers
Editorial Board Member 2003 - 2004

ChemKnowledge Micromedex

Editorial Advisory Board 2002 - 2006

TOMES Editorial Board
Senior Associate Editor/Toxicology Medical Advisor 2000 - 2006
Associate Editor: 1996 - 2000

POISINDEX Editorial Board
Senior Associate Editor/Toxicology Medical Advisor 2000 - 2006

EDITORIAL ACTIVITIES - TEXTBOOKS

1. Occupational, Industrial and Environmental Toxicology. Greenberg, Hamilton, Phillips, eds. Mosby-Year Book Publishers, St. Louis, MO, 1997.

2. Occupational, Industrial and Environmental Toxicology. (Second Edition) Greenberg, Hamilton, Phillips, McCluskey, eds. Mosby Publishers, St. Louis, MO, 2003.

3. Clinics in Occupational and Environmental Medicine: Terrorism: Biological, Chemical and Nuclear. Chase, Upfal, Krieger, Phillips, Guidotti, Weissman, eds. Vol. 2, No. 2, Saunders Div. of Elsevier Science. Orlando, FL, 2003.

4. Scientific Literature Review of Mold: A report on the health effects of indoor mold. National Association of Home Builders. Washington, DC., September 2003.

5. Industrial Solvents and Human Health, Part I. Phillips SD, Krieger GR, eds.. Elsevier Saunders Pub., Philadelphia PA, August 2004. (Clinics in Occupational and Environmental Medicine, Vol. 4, No. 3.)

6. Industrial Solvents and Human Health, Part II. Phillips SD, Krieger GR, eds.. Elsevier Saunders Pub., Philadelphia PA, November 2004. (Clinics in Occupational and Environmental Medicine, Vol. 4, No. 4.)

7. Critical Care Toxicology: Diagnosis and Management of the Critically Poisoned Patient. Brent, Wallace, Burkhart, Phillips, Donovan, eds. Elsevier Mosby Publishers. Philadelphia, PA. 2005.

8. Occupational and Environmental Medicine Review. Pearls of Wisdom. Greenberg, Hendrickson, Lyons, Madsen Meier, Morocco, Phillips, Waksman, eds. McGraw Hill Medical Publishing Division, New York City, NY. 2006.

9. Medical Toxicology of Drug Abuse. Synthesized Chemicals and Psychoactive Plants. Barceloux Ed. Editorial Review Panel. Wiley Hoboken, NJ 2012.

10. Clinical Practice of Biological Monitoring. Hoffman, Palmer, Phillips, Eds. OEM Press, Beverly Farms, MA 2012.

11. Clinical Practice of Biological Monitoring. eBook Hoffman, Palmer, Phillips, Eds. OEM Press, Beverly Farms, MA 2013.

PEER REVIEWER for:

Reviewer: Critical Reviews in Toxicology	2008-Present
Reviewer: Journal of Medical Toxicology	2006-Present
Reviewer: Journal of the American Medical Association	2007-Present
Reviewer: Archives of Environmental and Occupational Health	2006 - Present
Reviewer: Journal of Agricultural Medicine	2004 - Present
Reviewer: Journal of Occupational & Environmental Medicine	2002 - Present
Reviewer: American Institute of Biological Sciences	2000 - Present

Reviewer: Journal of Toxicology Clinical Toxicology	2000 - Present
Reviewer: International Journal of Toxicology	1999 - Present
Reviewer: Archives of Internal Medicine	1995 - Present
Reviewer: Annals of Emergency Medicine	1993 - Present
Reviewer: Journal Emergency Medicine	1994 - Present
Reviewer: Agency for Toxic Substances Disease Registry	1993 - Present
Editorial Board: Rocky Mountain Poison Center Bulletin	1992 - 1994
Reviewer: The Journal of Critical Illness	1988 – 1992
Abstract Reviewer: North American Congress of Clinical Toxicology	1992 - 2000

INVITED LECTURES

October 1, 1990. Emergency Treatment of Methemoglobinemia. St. Joseph's Hospital Medical Center, Denver, CO.

October 9, 1990. Environmental Toxins. Emergency Medicine Conference, Denver General Hospital, Denver, CO.

October 26, 1990. Digitalis Toxicity. University of Colorado Health Sciences Center, Clinical Pharmacology/Clinical Toxicology Conference, Denver, CO.

November 3, 1990. Responding to Poisoning Emergencies, Family Practice Update, AMI/Presbyterian-Saint Luke's Medical Center, Denver, CO.

November 15, 1990. Role of Poison Centers in the Health Care System, Univ. CO Health Sci Center, School of Pharmacy, Denver, CO.

January 4, 1991. Analgesic Toxicity: Recognition and Response, Saint Luke's Medical Center, Denver, CO.

February 5, 1991. Treatment of Anticholinergic Poisoning. Emergency Medicine Conference, Denver General Hospital, Denver, CO.

May 3, 1991. Unknown Toxicological Emergency Medical Grand Rounds, Riverton Memorial Hospital, Riverton, WY.

May 24, 1991. Mushroom Poisoning and Management, Internal Medicine Conference. Framingham Union Hospital, Framingham, MA.

June 4, 1991. Mushroom Poisoning and Management, Emergency Medicine Conference, Denver General Hospital, Denver, CO.

June 18, 1991. Acute Methanol Poisoning in Children and Treatment, The Children's Hospital Pediatric Conference, Denver, CO.

June 26, 1991. Clandestine Laboratory Health Hazards, New Mexico State Police, FBI and DEA Training Course, Santa Fe, NM.

August 28, 1991. Clandestine Laboratory Health Hazards. Wyoming State Police, FBI and DEA, Cheyenne, WY.

September 4, 1991. Pediatric Gastrointestinal Decontamination. Pediatric Conference. Fitzsimons's Army Medical Center, Denver, CO.

September 13, 1991. Recognition and Treatment of Antidepressant Poisoning. Clinical Conference, Littleton-Porter Hospital, Littleton, CO.

November 6, 1991. Arthropod Envenomation's, Conf. at Rocky Mtn Poison Center, Denver, CO.

November 19, 1991. The Management of Toxicological Emergencies, Toxicology Conference at The Blodgett Regional Poison Center, Blodgett Memorial Hospital, Grand Rapids, MI.

December 3, 1991. Snake Venom Poisoning in North America, Emergency Dept Lecture Series, Denver General Hospital, Univ. of Colorado Health Sciences Center, Denver, CO.

January 22, 1992. Emergency approach to the Toxic Patient, Emergency Dept Lecture Series, Univ. Colorado Health Sciences Center, Denver, CO.

January 31, 1992. Arthropod Venom Poisoning, Pathophysiology of Disease Course, 2nd year Class, Univ. Co Health Sciences Center, Denver, CO.

February 19, 1992. Tricyclic Antidepressant Poisoning, Rocky Mtn Poison Center, Denver General Hospital, Univ. Co Health Sciences Center, Denver, CO.

February 22, 1992. Environmental Health Toxic Substances that Affect Our Health. Metropolitan State College of Denver, Alumni Workshop, Denver, CO.

May 6, 1992. Ocular Acid and Base Toxicity and Current Research in Treatment, Rocky Mtn Poison Center, Denver, CO.

May 7, 1992. Gastrointestinal Decontamination - Toxicology Conference at Humana-Sunrise Hospital, Las Vegas, NV.

May 7, 1992. Tricyclic Antidepressant Toxicity - Toxicology Conference at Humana-Sunrise Hospital, Las Vegas, NV.

May 7, 1992. Bugs, Bites and Stings- Toxicology Conference at Humana-Sunrise Hospital, Las Vegas, NV.

May 11, 1992. Brown Spider Envenomation - A trial of three Therapies. UCHSC - Emergency Medicine Research Symposium, University of Colorado Health Sciences Center, Denver, CO.

May 13, 1992. The Role of Regional Poison Centers in Animal Poisoning: Where do we go from here? Rocky Mtn Poison Center Veterinary Toxicology Conference "Pets and Poisons", Denver, CO.

July 13, 1992. Toxic Alcohols: Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

July 31, 1992. Reptile Envenomation's. Swedish Medical Center Paramedic In-service Conference.

August 3, 1992. Anti-arrhythmics: Poisoning and Profiles of Action: Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

September 21, 1992. Brown Spider Envenomation: A study of Three Therapies Platform Presentation, 1992 AAPCC, ABMT, AACT, CAPCC Annual Meeting in Tampa, FL.

October 16, 1992. Arthropod Venom Poisoning: Swedish Medical Center Paramedic In-service Conference.

October 26, 1992. Mushroom Poisoning: Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

October 30, 1992. Necrotic Arachnidism: Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

January 27, 1993. Brown Spider Envenomation: A Study of Three Therapies, Winter Symposia on Hyperbaric Medicine, Steamboat Springs, CO.

July 20, 1993. Arthropod Venom Poisoning, Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

September 14, 1993. Cutaneous Arachnidism, Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

September 27, 1993. Antidepressant Poisoning, Porter Memorial Hospital, "Toxicology Symposia", Denver, CO.

September 27, 1993. Hazardous Materials Toxicology, Porter Memorial Hospital, "Toxicology Symposia", Denver, CO.

September 30, 1993. The Hazards of Hypnotics, Golden, CO.

October 25, 1993. Sleep Pharmacology, The Hazards of Hypnotics, Medical Grand Rounds, University of Colorado Health Sciences Center, Denver, CO.

February 15, 1994. Carbon Monoxide Poisoning and the Mental Status Exam, Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

March 12, 1994. Snake Venom Poisoning, Black Hills Winter Conference on Emergency Medicine, Rapid City, SD.

March 12, 1994. Case Studies in Toxicology (hydrogen sulfide, carbon monoxide, chlorine gas), Black Hills Winter Conference on Emergency Medicine, Rapid City, SD.

September 25, 1994. Fluoxetine v. Tricyclic Antidepressants: A Prospective Multi-center Trial. North American Congress of Clinical Toxicology (NACCT), Salt Lake City, UT.

October 5, 1994. Antidepressant Poisoning, Annual Toxicology Conference, Porter Memorial Hospital, Denver, CO.

October 5, 1994. Antiarrhythmic Poisoning, Annual Toxicology Conference, Porter Memorial Hospital, Denver, CO.

October 19, 1994. Hyperbaric Oxygen Therapy in Carbon Monoxide Poisoning, Lutheran Medical Center Emergency Department, Wheat Ridge, CO.

October 25, 1994. Seminar Director: Toxicology Emergencies in the Workplace: Preparation and Management ACOEM State-of-the-Art Conference, Denver, CO.

October 25, 1994. Hyperbaric Oxygen Therapy in Cellular Poisonings. ACOEM State-of-the-Art Conference, Denver, CO.

October 27, 1995. Toxic Case Presentations (hydrogen sulfide, carbon monoxide, chlorine and ammonia gas), ACOEM State-of-the-Art, Denver, CO.

June 27, 1995. Hazardous Waste/Materials (cyanide, hydrogen sulfide and carbon monoxide), National Environmental Health Association Annual Meeting. Session Director, Denver, CO.

June 27, 1995. Heavy Metal Poisoning: Cadmium, Lead, Mercury National Environmental Health Association Annual Meeting, Denver, CO.

June 27, 1995. Risk Communication, National Environmental Health Association Annual Meeting, Denver, CO.

July 27-28, 1995. Medical Monitoring Programs, Rocky Mountain Environmental Health and Safety Forum.

September 5, 1995. Organophosphate Poisoning. Rose Medical Center Internal Medicine Mortality and Morbidity Conference, Denver, CO.

September 15, 1995. Acute Tricyclic Antidepressant Poisoning. Grand Rounds in Emergency Medicine, Medical College of Pennsylvania, Philadelphia, PA.

September 28, 1995. Explosives Toxicology: Remnants in Soil and Water. Agency for Toxic Substances & Disease Registry of Centers for Disease Control & St. Francis Medical Center, Grand Isle NE.

October 3, 1995. Acute Tricyclic Antidepressant Poisoning. Grand Rounds in Internal Medicine, MetroWest Medical Center, Framingham, MA.

October 24, 1995. Health Effects of Soil and Water Contamination. ACOEM 1995 State-of-the-Art Conference, Seattle, WA.

December 13, 1995. Use of 4-Methylpyrazole in Methanol and Ethylene Glycol Poisoning. Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

January 15, 1996. Baromedicine and Carbon Monoxide Poisoning. PorterCare Hospital, Denver, CO.

January 19, 1996. Cadmium Poisoning in the Workplace. Rocky Mountain ACOEM 31st Annual Conference on Occupational Medicine, Denver, CO.

January 19, 1996. Cyanide Poisoning Cases from Local Industry. Rocky Mountain ACOEM 31st Annual Conference on Occupational Medicine, Denver, CO.

February 20, 1996. Toxic Alcohols and Treatment. The Children's Hospital, Denver, CO.

February 22, 1996. Ethylene Glycol, Methanol, and Isopropanol Poisoning. Denver General Hospital, Denver, CO.

March 4, 1996. Health Hazards of Water Pollution, Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

July 2, 1996. Chemical Carcinogenesis, National Environmental Health Association 60th Annual Meeting, Chicago, IL.

July 2, 1996. Radon, National Environmental Health Association 60th Annual Meeting, Chicago, IL.

July 2, 1996. Heavy Metal Carcinogenesis, National Environmental Health Association 60th Annual Meeting. Chicago, IL.

July 2, 1996. Investigating Cancer Clusters, National Environmental Health Association 60th Annual Meeting. Chicago, IL.

September 30, 1996. Chemical Carcinogenesis, Rocky Mountain Poison Center, University of Colorado Health Sciences Center, Denver, CO.

October 22, 1996. Salicylate Poisoning, The Children's Hospital, University of Colorado Health Sciences Center, Denver, CO.

June 29, 1997. Organophosphate Toxicity, National Environmental Health Association 61st Annual Meeting, Washington, DC.

June 29, 1997. Environmental Monitoring of Organophosphates, National Environmental Health Association 61st Annual Meeting, Washington, DC.

June 29, 1997. Cancer Clusters, National Environmental Health Association 61 Annual Meeting, Washington, DC.

July 9, 1997. Beta-Blocker and Calcium Channel Blocker Poisoning. Rocky Mountain Poison and Drug Center, University of Colorado Health Sciences Center, Denver, CO.

July 16, 1997. Carbon Monoxide, Cyanide and Hydrogen Sulfide Poisoning. Rocky Mountain Poison and Drug Center, University of Colorado Health Sciences Center, Denver, CO.

September 15, 1997. The Utility of Magnetic Resonance Imaging in the Bite from the Prairie Rattlesnake. North American Congress of Clinical Toxicology Annual Meeting, St. Louis, MO.

January 16, 1998. Low Level Toxicants and Causation. The Rocky Mountain Academy of Occupational & Environmental Medicine and Denver Colorado Association of Occupational Health Nurses. The 33rd Annual Conference on Occupational Medicine, Health & Safety, Denver, CO.

July 1, 1998. Basic Toxicology Course. National Environmental Health Association 62nd Annual Meeting, Las Vegas, NV.

October 22, 1998. Limitations of the MSDS. Toxicology Information Roundtable TIR' 98, Denver, CO.

February 19, 1999. Antidote Stocking. Ada County Medical Society Annual Medical Conference, McCall, ID.

May 20, 1999. Cotton Dust Exposure. Rocky Mountain Poison and Drug Center, University of Colorado Health Sciences Center, Denver, CO.

July 6, 1999. Practical Toxicology for Environmental Health Specialists. National Environmental Health Association, Nashville, TN.

October 2, 1999. Volatile Organic Compounds, and Drinking Water Regulations. American Academy of Clinical Toxicology Occupational and Environmental Symposium at the North American Congress of Clinical Toxicology Annual Meeting, La Jolla, CA.

October 18, 1999. Workshop on Plasticizers: Scientific Issues in Blood Collection, Storage and Transfusion. Panel Discussion. Food and Drug Administration, National Institutes of Health, Bethesda, MD.

October 26, 1999. The Role of Physicians in Community Right-to-Know. 1999 NEHA Right-to-Know Conference and Exhibition, Denver, CO.

October 27. 1999. Understanding How the Environment Impacts Public Health: Information Needs of Health Care Providers. 1999 NEHA Right-to-Know Conference and Exhibition, Denver, CO.

December 9, 1999. Clinical Aspects of Medical Monitoring and Medial Surveillance. National Medical Services, Willow Grove, PA.

December 10, 1999. Selected Cases in Toxicology. Medical College of Pennsylvania-Drexel University, Philadelphia, PA.

December 10, 1999. 4-Methylpyrazole as an Antidote for Toxic Alcohols. Medical College of Pennsylvania-Drexel University, Philadelphia, PA.

February 11, 2000. Precautionary Principle from the Medical Toxicologists Perspective. The Toxicology Forum, Washington D.C.

May 15, 2000. Occupational Toxicology: A Historical Perspective. American College of Occupational & Environmental Medicine: American Occupational Health Conference, Philadelphia, PA.

June 16, 2000. Toxicology and Exposure Assessment. National Environmental Health Association, Denver, CO.

September 15, 2000. Medical Toxicology Fellows Opportunities in Occupational and Environmental Toxicology Seminar. 2000 North American Congress of Clinical Toxicology, Tucson, AZ.

September 17, 2000. The Role of the Medical Toxicologist in Consulting with Industry an Hour in the Life. 2000 North American Congress of Clinical Toxicology, Tucson, AZ.

September 19, 2000. Diesel Fuel and Diesel Exhaust Toxicology. American Industrial Hygiene Association/American Society of Safety Engineers Fall 2000 Technical Conference. Colorado School of Mines, Golden, CO.

December 8, 2000. Internal Medicine M & M Conference. University of Colorado Health Sciences Center, Denver, CO.

April 26, 2001. Introduction to Toxicology Principles. Farmworker Children, Health and the Environment: A Pediatric Environmental Health Intensive. 2001 National Farmworker Health Conference, San Juan, Puerto Rico.

April 26, 2001. Water and Sanitation Session, Toxicologic Aspects of Drinking Water Contamination. Farmworker Children, Health and the Environment: A Pediatric Environmental Health Intensive. 2001 National Farmworker Health Conference, San Juan, Puerto Rico.

April 26, 2001. Pesticide Toxicology. Farmworker Children, Health and the Environment: A Pediatric Environmental Health Intensive. 2001 National Farmworker Health Conference, San Juan, Puerto Rico.

May 2, 2001. Risk Communication and Toxicology. University of Colorado Health Sciences Center, Rocky Mountain Poison and Drug Center, Denver, CO.

June 23, 2001. Pesticides and Human Health. Clinical & Environmental Issues on the Border. Toxicology has No Borders. Fourth Bi-National Conference. Texas Tech health Sciences Center- Emergency Medicine. University of Texas at El Paso-CERM & HETCAT, El Paso, TX.

June 27, 2001. The Use of the Internet as a Clinical and Scientific Information Source. Colorado Council of Medical Librarians. Porter Adventist Hospital, Denver, CO.

October 8, 2001. National Library of Medicine Workshop: Web Resources in Clinical Toxicology. A Medical Toxicologist Perspective on Web-Based Toxicology Resources. North American Congress of Clinical Toxicology Annual Meeting, Montreal, Quebec, Canada.

October 9, 2001. American Academy of Clinical Toxicology – Year in Toxicology. Systemic Lupus Erythematosus. North American Congress of Clinical Toxicology Annual Meeting, Montreal, Quebec, Canada.

October 9, 2001. Topics in Industrial Hygiene, Environmental Monitoring, and Exposure Assessment for Clinical Toxicologists. How dose and Exposure Become a Dose? North American Congress of Clinical Toxicology Annual Meeting, Montreal, Quebec, Canada.

October 31, 2001. Update of Biological and Chemical Terrorism: Clinical Presentation, Management, and Preparedness. American College of Occupational and Environmental Medicine State-of-the-Art Conference, Seattle, WA.

December 12, 2001. Industry Preparation for Nuclear Biological and Chemical Events. IPE Industry Conference, Englewood, CO.

December 14, 2001. Standing Tall: Emergency Preparedness Training for Business. Medical Facts and Issues. South Metro Denver Chamber of Commerce, Centennial, CO.

March 4, 2001. Environmental Toxicology. Toxicology Grand Rounds MicroMedix, Thompson Publishing, Englewood, CO.

April 14, 2002. Introduction and Overview of Neurotoxicology. Millennium Series Occupational & Environmental Toxicology session on Clinical Neurotoxicology. 2002 American Occupational Health Conference, ACOEM, Chicago, IL.

September 29, 2002. A Medical Toxicologists Perspective on Web-based Toxicology Resources. National Library of Medicine Symposium: digital Resources for Clinical Toxicology. 2002 North American Congress of Clinical Toxicology, Palm Springs, CA.

October 10, 2002. Selected Toxicology Case Presentations. Deaconess Medical Center, Spokane, WA.

October 10, 2002. Common Cardiovascular Toxicants. Deaconess Medical Center, Spokane, WA.

October 27, 2002. Fertilizer and Chemical Toxicology. 2002 State of the Art Conference Millennium Series, Agrochemical Toxicology. American College of Occupational Environmental Medicine, Baltimore, MD.

October 27, 2002. Agrochemical Toxicology. 2002 State of the Art Conference Millennium Series, Agrochemical Toxicology. American College of Occupational Environmental Medicine, Baltimore, MD.

June 16, 2003. Clinical Aspects of Pesticide Intoxication: Pesticides & Sovereignty. Pesticide Illness Prevention Management and Enforcement on the Flathead Reservation, Polsun, MT.

June 16, 2003. Bioterrorism, Homeland Preparedness and Pesticides: Pesticides & Sovereignty. Pesticide Illness Prevention Management and Enforcement on the Flathead Reservation, Polsun, MT.

June 16, 2003. Special Session: Pesticide Toxicity and Health Care Providers: Pesticides & Sovereignty. Pesticide Illness Prevention Management and Enforcement on the Flathead Reservation, Polsun, MT.

July 28, 2003. Overview and introduction to pesticides and prevention: Pesticide Intoxication. Recognition, Management and Prevention in Pacific Northwest Tribal Agriculture, Nespelem, WA.

July 28, 2003. Obtaining an Occupational and Environmental Exposure History: Pesticide Intoxication. Recognition, Management and Prevention in Pacific Northwest Tribal Agriculture, Nespelem, WA.

July 28, 2003. Bioterrorism and Pesticide disease surveillance systems in high risk populations: Pesticide Intoxication. Recognition, Management and Prevention in Pacific Northwest Tribal Agriculture, Nespelem, WA.

July 28, 2003. Implication of research on insecticide and other pesticides found in farm workers or high risk populations: Pesticide Intoxication. Recognition, Management and Prevention in Pacific Northwest Tribal Agriculture, Nespelem, WA.

July 30, 2003. Exposure Assessment for the Primary Health Care Provider, Firefighting and Pesticide-related Illness. Recognition, Management and Prevention of asthma and Pesticide Illnesses: A course for health care professionals, environmental professionals, emergency responders, school leaders and Nez Perce community member, Lapwai, ID.

July 30, 2003. Responses to Bioterrorism: Focus on Pesticide Chemicals an other weapons of Terrorism. Recognition, Management and Prevention of asthma and Pesticide Illnesses: A course for health care professionals, environmental professionals, emergency responders, school leaders and Nez Perce community member, Lapwai, ID.

September 8, 2003. National Library of Medicine Symposium: Using the Web to Access Clinical Toxicology Information. A Medical Toxicologists Perspective on Web Resources. North American Congress of Clinical Toxicology, Chicago, IL.

July 29, 2004. Pulmonary Inhalation Injuries. Rocky Mountain Poison and Drug Center, University of Colorado Health Sciences Center, Denver, CO.

September 13, 2004. National Library of Medicine Symposium: Using the Web to Access Clinical Toxicology Information. A Medical Toxicologists Perspective on Web Resources. North American Congress of Clinical Toxicology, Seattle, WA.

September 22, 2004. Disinfectant Byproducts the Clinical Perspective. Disinfection Byproducts Symposium. CIIT Centers for Health Research. Research Triangle Park, NC.

September 29, 2004. Observed Behaviors During Mass Chemical Exposures: Are All of These Patients Poisoned? Public Health in the Rocky Mountains 2004. Weapons of Opportunity. Breckenridge, CO.

September 29, 2004. Chemical Terrorism of Food and Water. Public Health in the Rocky Mountains 2004. Weapons of Opportunity. Breckenridge, CO.

October 20, 2004. Chemical Agents of Opportunity for Terrorism. The Medical and Psychological Consequences of TICs (Toxic Industrial Chemicals) and TIMs

(Toxic Industrial Materials). American College of Medical Toxicology/Agency for Toxic Substances Disease Registry. Montana Department of Health and Human Services. Helena, MT.

February 8, 2005. Chemical Agents of Opportunity for Terrorism. The Medical and Psychological Consequences of TICs (Toxic Industrial Chemicals) and TIMs (Toxic Industrial Materials). Agrochemical Terrorism. American College of Medical Toxicology/Agency for Toxic Substances Disease Registry. US Environmental Protection Agency. Agency for Toxic Substance Disease Registry. Las Vegas, NV.

May 17, 2005. Vapor Intrusion from Groundwater Contamination. Davis Community Hospital, Layton, UT.

May 18, 2005. Vapor Intrusion from Groundwater Contamination. Restoration Advisory Board. Weber State-Layton campus, Layton, UT.

March 3, 2006. Introduction to Medical Toxicology. Presbyterian St. Lukes Hospital. Pediatric Grand Rounds, Denver CO.

April 2nd , 2006. Toxicology Principles. National Jewish Medical and Research Center. Denver, CO.

April 16th , 2006. Basic Occupational Toxicology. National Jewish Medical and Research Center. Denver, CO.

April 28th , 2006. Environmental Toxicology. National Jewish Medical and Research Center. Denver, CO.

August 7th, 2006. Terrorism by Fear and Uncertainty: Delayed Toxic Syndromes. Chemical Agents of Opportunity for Terrorism: The Medical Consequences of TICs (Toxic Industrial Chemicals) and TIMs (Toxic Industrial Materials). 9th Annual Forces Health Protection Conference CHIPPM. Albuquerque, NM.

October 8, 2006. AACT Year in Toxicology: The New OSHA Chromium Standard. North American Congress of Clinical Toxicology, Annual Meeting. San Francisco, CA.

February 9, 2007. Introduction to Pulmonary Toxicology. Pulmonary and Critical Care Fellows, University of Colorado Health Sciences Center, Veterans Affairs Medical Center, Denver, Co.

February 23rd, 2007. Management of the Acutely Poisoned Patient. Pulmonary and Critical Care Fellows, University of Colorado Health Sciences Center, Veterans Medical Center, Denver, CO

March 6th, 2007. Introduction to Medical Toxicology. HealthOne Outreach Lectures. Miners' Colfax Medical Center, Raton, NM

May 31, 2007. Public Health Impact Assessments and Toxicology in Vietnam. BP Health Impact Assessment Workshop, Boulder, Co.

August 2, 2007. Inhalation Toxicology. Rocky Mountain Poison & Drug Center, University of Colorado Health Sciences Center. Denver, CO

August 5, 2007. Chemical Contamination of Food, Drinks and Drugs. Chemical Agents of Opportunity for Terrorism: The Medical Consequences of TICs (Toxic Industrial Chemicals) and TIMs (Toxic Industrial Materials). 10th Annual Forces Health Protection Conference CHIPPM. Louisville, KY.

September 14, 2007. Biomonitoring of Hazardous Materials in the Workplace. Medichem International Congress XXXV. Queretaro, Mexico.

September 20, 2007. Introduction to Occupational and Environmental Toxicology. National Jewish Medical and Research Center. Denver, CO.

September 24, 2007. Methamphetamine Production & Abuse on Tribal Lands. The National Indian Health Board, 24th Annual Conference. Portland, OR.

October 25th, 2007. Medical Surveillance. Basic Curriculum in Occupational Medicine. State-of-the-Art Conference. American College of Occupational & Environmental Medicine. Vancouver, BC.

October 25th, 2007. Respiratory Protection. Basic Curriculum in Occupational Medicine. State-of-the-Art Conference. American College of Occupational & Environmental Medicine. Vancouver, BC.

November 27th, 2007. Toxicology Concepts in Public Health. National Jewish Medical and Research Center. Denver, CO.

March 13th, 2008. Toxicology and Vapor Intrusion. Hill AFB RAB, Roy, UT

July 12th, 2008. Cardiology Update. Basic Curriculum in Occupational Medicine. State-of-the-Art Conference. American College of Occupational & Environmental Medicine. Chicago, Il.

July 12th, 2008. Hematology Update. Basic Curriculum in Occupational Medicine. State-of-the-Art Conference. American College of Occupational & Environmental Medicine. Chicago, Il.

July 17-18th, 2008. The Clinical Neurotoxicology of Chemical Terrorism & Terrorism by Fear and Uncertainity: Delayed Toxic Syndromes. Chemical Agents of Opportunity for Terrorism: The Medical Consequences of TICs (Toxic Industrial Chemicals) and TIMS (Toxic Industrial Materials). Tumon, Guam.

August 10th, 2008. Toxic Gases in your Community & Cyanide and Fumigants. Chemical Agents of Opportunity for Terrorism: The Medical Consequences of TICs (Toxic Industrial Chemicals) and TIMS (Toxic Industrial Materials). 11th Annual Forces Health Protection Conference CHIPPM Albuquerque, NM.

September 15th, 2008. The Practice of Outpatient Occupational and Environmental Toxicology. ACMT Practice Symposium: New Dimensions in the Practice of Medical Toxicology. North American congress of Clinical Toxicology 2008, Toronto, Canada

November 5th, 2008. Intralipid Therapy in Toxicology. Vietnam National Toxicology Conference, Hanoi, Vietnam.

November 6th, 2008. Renal Toxicology. Vietnam National Toxicology Conference, Hanoi, Vietnam.

Janurary 24th, 2009. Occupational & Enviornamental Toxicology. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

February 14th, 2009. Reproductive and Developmental Toxicology. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

February 21st, 2009. Toxicology of Vinyl Chloride. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

March 7th, 2009. Toxicology of the Skin. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

March 21st, 2009. Endocrine Toxicology. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

March 21st, 2009. Toxicology Review. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

April 18th, 2009. Aquatic Toxicology and Water Pollution. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

May 2nd, 2009. Health Impact Assessments. International Emerging Toxicology. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

May 4th, 2009. Toxicology Review. Environmental and Occupational Toxicology (PMD 6616), University of Colorado Denver. Aurora, Colorado.

May 25th , 2009. Occupational & Environmental Toxicology. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

May 26th, 2009. Pattern Recognition and Toxic Syndromes. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

May 27th, 2009. Carbon Monoxide. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

May 28th, 2009. Mechanistic Toxicology: Kinetics of poisons, exposure, and risk. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

June 2th, 2009. Mechanistic Toxicology: Kinetics of poisons, exposure, and risk. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

June 3th, 2009. Occupational & Environmental Toxicology. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

June 3th, 2009. Pattern Recognition and Toxic Syndromes. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

June 4th, 2009. Pulmonary Toxicology. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

June 4th, 2009. Carbon Monoxide. WHO Fellowship Lecture Program. Bach Mai Hospital, Hanoi Vietnam.

November 21, 2009. Introduction to Toxicology. ACOEM Basic Curriculum course. Cincinnati, OH.

April 21, 2010. Occupational Toxicology. Western Medical Toxicology Fellows Meeting. Denver, CO

November 6th, 2010. Toxicology Principles. ACOEM Basic Curriculum in Occupational Medicine. Seattle, WA

November 6th, 2010. Health Surveillance. ACOEM Basic Curriculum in Occupational Medicine. Seattle, WA

November 6th, 2010. Respirator Clearance. ACOEM Basic Curriculum in Occupational Medicine. Seattle, WA

March 24th, 2011. Cardiology. ACOEM Basic Curriculum in Occupational

Medicine. Washington, DC

March 24th, 2011. Hematology. ACOEM Basic Curriculum in Occupational Medicine. Washington, DC

March 25th, 2011. Neurotoxicity. ACOEM Basic Curriculum in Occupational Medicine. Washington, DC

March 24th, 2011. Reproductive Toxicology. ACOEM Basic Curriculum in Occupational Medicine. Washington, DC

March 24th, 2011. Metal Toxicology. ACOEM Basic Curriculum in Occupational Medicine. Washington, DC

November 12th, 2011. Health Surveillance. ACOEM Foundations of Occupational Medicine. Denver, CO

November 12th, 2011. Respirator Clearance. ACOEM Foundations of Occupational Medicine. Denver, CO

November 12th, 2011. Toxicology Principles. ACOEM Foundations of Occupational Medicine. Denver, CO

April 27th, 2012. Cardiology. ACOEM Foundations of Occupational Medicine. Los Angeles, CA

April 27th, 2012. Hematology. ACOEM Foundations of Occupational Medicine. Los Angeles, CA

April 27th, 2012. Neurotoxicology. ACOEM Foundations of Occupational Medicine. Los Angeles, CA

April 27th, 2012. Reproductive. ACOEM Foundations of Occupational Medicine. Los Angeles, CA

April 27th, 2012. Metal Toxicology. ACOEM Foundations of Occupational Medicine. Los Angeles, CA

August 7th, 2012. Enhancing Health Impact Assessment Methodologies for Polar Communities. 15th International Congress On Circumpolar Health. Fairbanks, AK

November 7th, 2012. Pediatric Lead Poisoning. Pediatric Medicine Symposium Bach Mai Hospital, Hanoi Vietnam

November 7th, 2012. Neonatal Toxicology. Pediatric Medicine Symposium Bach Mai Hospital, Hanoi Vietnam

April 26th, 2013. Cardiology. ACOEM Foundations of Occupational Medicine. Orlando, FL

April 26th, 2013. Hematology. ACOEM Foundations of Occupational Medicine. Orlando, FL

April 27th, 2013. Neurotoxicology. ACOEM Foundations of Occupational Medicine. Orlando, FL

April 27th, 2013. Reproductive. ACOEM Foundations of Occupational Medicine. Orlando, FL

April 27th, 2013. Metal Toxicology. ACOEM Foundations of Occupational Medicine. Orlando, FL

November 7th, 2013. Arsenic Toxicology. International Conference on Medical Toxicology. Hanoi, Vietnam

November 8th, 2013. Food Additives and Contamination. International Conference on Medical Toxicology. Hanoi, Vietnam

November 8th, 2013. Mycotoxins in Food. International Conference on Medical Toxicology. Hanoi, Vietnam

January 24th, 2014. Neurobiology & Pharmacotherapy of Alcohol Withdrawal. Delta County Memorial Hospital, Delta, CO

April 25th, 2014. Neurotoxicity. ACOEM Foundations in Occupational Medicine. American College of Occupational Environmental Medicine, San Antonio, TX

April 25th, 2014. Reproductive Health. ACOEM Foundations in Occupational Medicine. American College of Occupational Environmental Medicine, San Antonio, TX

April 25th, 2014. Hematology In Occupational Medicine. ACOEM Foundations in Occupational Medicine. American College of Occupational Environmental Medicine, San Antonio, TX

April 25th, 2014. Cardiology in Occupational Medicine. ACOEM Foundations in Occupational Medicine. American College of Occupational Environmental Medicine, San Antonio, TX

April 25th, 2014. Metal Toxicology. ACOEM Foundations in Occupational Medicine. American College of Occupational Environmental Medicine, San Antonio, TX

October 18th, 2014. Preventing ICU Complications Prophylactic Measures for the Poisoned Patient. American Academy of Clinical Toxicology Pre-symposium Meeting. North American Congress for Clinical Toxicology. New Orleans, LA

November 9th, 2014. Introduction to Toxicology. ACOEM Foundations of Occupational Medicine. San Diego, CA

November 9th, 2014. Medical Surveillance. ACOEM Foundations of Occupational Medicine. San Diego, CA

November 9th, 2014. Respiratory Clearance. ACOEM Foundations of Occupational Medicine. San Diego, CA

April 16th, 2015. Preventative Therapeutics in Critical Care. International Critical Care symposium, Da Nang, Vietnam

April 16th, 2015. Critical care Toxicology Update. International Critical Care symposium, Da Nang, Vietnam

April 16th, 2015. Critical Care Pharmacology Update. International Critical Care symposium, Da Nang, Vietnam

May 2nd, 2015. Introduction to Toxicology. American College of Occupational Environmental Medicine. (ACOEM) American Occupational Health Conference. Foundations in Occupational Medicine, Baltimore, MD

May 2nd, 2015. Neurotoxicology. American College of Occupational Environmental Medicine. (ACOEM) American Occupational Health Conference. Foundations in Occupational Medicine, Baltimore, MD

May 2nd, 2015. Reproductive & Developmental Toxicology. American College of Occupational Environmental Medicine. (ACOEM) American Occupational Health Conference. Foundations in Occupational Medicine, Baltimore, MD

May 2nd, 2015. Cardiovascular and Hematological Toxicology. American College of Occupational Environmental Medicine. (ACOEM) American Occupational Health Conference. Foundations in Occupational Medicine, Baltimore, MD

May 2nd, 2015. Hepatic and Renal Toxicology. American College of Occupational Environmental Medicine. (ACOEM) American Occupational Health Conference. Foundations in Occupational Medicine, Baltimore, MD

May 2nd, 2015. Pesticide Toxicology. American College of Occupational Environmental Medicine. (ACOEM) American Occupational Health Conference. Foundations in Occupational Medicine, Baltimore, MD

May 2nd, 2015. Metal Toxicology 1 and 2. American College of Occupational Environmental Medicine. (ACOEM) American Occupational Health Conference. Foundations in Occupational Medicine, Baltimore, MD

January 5th, 2016. Neurochemistry and Management of Alcohol Withdrawal. Providence Mount Carmel Hospital, Colville, WA

April 10th, 2016 Introduction to Medical Toxicology, Foundations in Occupational Medicine, American College of Occupation and Environmental Medicine (ACOEM-SOTC), Chicago, IL.

April 10th, 2016 Neurotoxicology, Foundations in Occupational Medicine, American College of Occupation and Environmental Medicine (ACOEM-SOTC), Chicago, IL.

April 10th, 2016 Syndromic Toxicology and Medical Practice. Foundations in Occupational Medicine, American College of Occupation and Environmental Medicine (ACOEM-SOTC), Chicago, IL.

April 10th, 2016 Toxicology of Metals, Foundations in Occupational Medicine, American College of Occupation and Environmental Medicine (ACOEM-SOTC), Chicago, IL.

April 10th, 2016 Clinical Dilemmas in Toxicology, Scientific Seminar on Emergency Medicine, Critical Care and Toxicology, Hanoi Vietnam

April 10th, 2016 Cardiovascular Toxicology In Critical Care, Scientific Seminar on Emergency Medicine, Critical Care and Toxicology, Hanoi Vietnam

April 10th, 2016 Hematological Toxicology in Critical Care, Scientific Seminar on Emergency Medicine, Critical Care and Toxicology, Hanoi Vietnam.

April 19th, 2016. Food Safety and Poisoning: natural and additives. Nha Trang, Vietnam

Meeting or Session Moderator

September 14, 1998. Basic Science Research, Platform Session 5, North American Congress of Clinical Toxicology Annual Meeting, Orlando, FL.

October 4, 1999. Keys to Success in the Managed Care Environment. American College of Medical Toxicology Practice Symposium. North American Congress of Clinical Toxicology Annual Meeting, La Jolla, CA.

October 4, 1999. Junk Science Attacks – Rescuing the Public from Media Hyped Toxic Scares: A Seminar in Risk Communication. North American Congress of Clinical Toxicology Annual Meeting, La Jolla, CA.

October 4, 1999. Keys to Success in the Managed Care Environment. North American Congress of Clinical Toxicology Annual Meeting, La Jolla, CA.

October 26, 1999. The Role of Health Care Providers in Providing Environmental Information and Interpretation of Data to the Public. 1999 NEHA Right-to-Know Conference and Exhibition, Denver, CO.

September 17, 2000. The Role of the Medical Toxicologist in Consulting with Industry an Hour in the Life. 2000 North American Congress of Clinical Toxicology, Tucson, AZ.

October 31, 2001. Update of Biological and Chemical Terrorism: Clinical Presentation, Management, and Preparedness. American College of Occupational and Environmental Medicine State-of-the-Art Conference, Seattle, WA.

April 14, 2002. Millennium Series Occupational & Environmental Toxicology session on Clinical Neurotoxicology. 2002 American Occupational health Conference, ACOEM, Chicago, IL.

September 29, 2002. NBC Boot Camp: Workshop on Biological Terrorism and Warfare Agents. 2002 North American Congress of Clinical Toxicology, Palm Springs, CA.

October 27, 2002. Agrochemical Toxicology. 2002 State of the Art Conference Millennium Series, Agrochemical Toxicology. American College of Occupational Environmental Medicine, Baltimore, MD.

February 8, 2005. Chemical Agents of Opportunity for Terrorism. The Medical and Psychological Consequences of TICs (Toxic Industrial Chemicals) and TIMs (Toxic Industrial Materials). Agrochemical Terrorism American College of Medical Toxicology/Agency for Toxic Substances Disease Registry. US Environmental Protection Agency. Agency for Toxic Substance Disease Registry. Las Vegas, NV.

March 28th, 2006. Introduction to Occupational Toxicology. National Jewish Medical and Research Center, University of Colorado Health Sciences Center, Denver, CO.

April 4th, 2006. Case Evaluations. National Jewish Medical and Research Center, University of Colorado Health Sciences Center, Denver, CO.

June 4-6th, 2009. Vietnam National Toxicology Symposium. Hanoi Vietnam.

November 7-8, 2013. International Conference on Medical Toxicology. Hanoi, Vietnam.

April 16-17th, 2015. International Critical Care Conference, Da Nang, Vietnam

April 15th, 2016. Critical Care Toxicology Symposium, Vietnam Society of Critical Care, Emergency Medicine and Toxicology. Hanoi Vietnam.

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10-10-16

LEGAL APPEARANCES*
By
SCOTT D. PHILLIPS, M.D., FACP, FACMT, FAACT

Date	Court Name	Parties Name	Case Number	Type of Testimony
9-9-10	District Court, County of Denver, Colorado	Krista Dawn Johnson v. HCA-Healthone, LLC d/b/a Rose Medical Center	No.: 09CV1985	Deposition
7-13-11	State of Louisiana 14TH Judicial District Court Parish of Calcaseiu	Tangela Brown, et al, vs. Georgia Gulf lake Charles, LLC	No.: 07-5068 B	Trial
10-12-11	District Court, County of Denver, State of Colorado	Krista Dawn Johnson v. HCA-Healthone, LLC d/b/a Rose Medical Center	No.: 09CV1985	Trial
2-17-12	Court of Common Pleas, State of South Carolina, County of Berkeley	Benjamin and Joy Allen vs D.R. Horton, Inc. et al	No.: 2008CP0802228A	Deposition
3-14-12	Court of Common Pleas, State of South Carolina, County of Berkeley	Benjamin and Joy Allen vs D.R. Horton, Inc. et al	No.: 2008CP0802228A	Trial
3-26-12	Division E, 14th Judicial District Court, Parish of Calcasieu, State of Louisiana	Edward Anthony, et al v. Georgia Gulf Lake Charles, LLC,	No.: 2007-5081	Deposition
6-15-12	Montana First Judicial District Court Lewis & Clark County	Angela Neff et al v. American Legion Institute of Family Living et al	No.: ADV-2010-694	Deposition

9-7-12	United States District Court Northern District of Indiana South Bend Division	CW, et al vs. Textron, Inc	No.: 3:10-CV-00087-PPS-CAN	Deposition
10-4-12	Colorado Denver County District Court	Moberley v. McDonalds	No.: 2012CV5715	Deposition
10-31-12	United States District Court for the Northern District of West Virginia – Clarksburg Division	Salvatore M. Bombardiere, Sr., v. Schlumberger Technology, et al	No.:1:11-cv-00050 (Bailey)	Deposition
3-8-13	United States District Court for the Northern District of West Virginia – Clarksburg Division	Salvatore M. Bombardiere, Sr., v. Schlumberger Technology, et al	No.:1:11-cv-00050 (Bailey)	Trial
3-27-13	State of South Dakota, County of Pennington. Circuit Court Seventh Judicial Circuit	State of South Dakota v Alfredo Vargas	No.: CR 12-3442	Hearing
8-29-13	District Court, Adams County, Colorado	M. Jeffrey Barger & Martha Cervantes v Trans-West et al	No.: 2011 cv 240	Deposition
9-19-13	State of South Dakota, County of Pennington. Circuit Court Seventh Judicial Circuit	State of South Dakota v Alfredo Vargas	No.: CR 12-3442	Trial

10-8-13	District Court, Adams County, Colorado	M. Jeffrey Barger & Martha Cervantes v Trans-West et al	No.: 2011 cv 240	Trial
1-17-14	Office of Administrative Hearings, State of Wyoming, County of Natrona	John F. Bucher v. State of Wyoming, ex rel., Department of Workforce Services, Workers' compensation	No.: 2013-00170	Deposition
3-13-14	Virginia Workers' Compensation Commission, Richmond, VA	Ryan Middleton v. FedEx Freight, Inc.	No.: JCN: VA 00000405924	Hearing
8-1-14	333rd Judicial District Court, Harris County, Texas	Roberto Gallegos, III et al , vs. Rig Runners, Inc, et al	No.: 2013-13945	Deposition
9-5-2014	District Court 3rd Judicial District, State of Idaho, County of Canyon	Eric P. Rangel, vs. Penny Wise Drug Stores Inc.	No.: 13-2166-C	Deposition

*These appearances have been prepared, and to the best of my knowledge, are all inclusive and accurate.